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PROLAPSE OF THE RECTUM

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INTRODUCTION AND CLASSIFICATION

PROLAPSE of the rectum is the abnormal presentation of part or all of the rectum through the anal orifice. It is classified as incomplete when only the mucosa prolapses, or complete when the entire rectum is involved; each may be mild, moderate or severe, depending upon its extent. High, or blind, prolapse of the rectum is telescoping of the rectosigmoid into the rectum which does not present through the anal orifice; this is strictly an intussusception and not a prolapse. Classification as first, second and third degree prolapse, depending upon the size or point of origin, as suggested by Bowers,[1] and other departures from this fundamental classification are confusing and should be avoided.

I. INCOMPLETE OR MUCOSAL PROLAPSE

An incomplete, simple, or mucosal prolapse of the rectum exists when a portion of the mucosa presents through the anal orifice due to abnormal mobility of its attachments. Redundancy of the mucosa usually coexists. In some instances constipation, diarrhea, polyps or hemorrhoids are associated with its appearance, but it has been assumed that, particularly in children, there is fundamentally a faulty union embryologically between the mucosa and the underlying structures at the anal margin where the hind gut and the proctoderm meet. Swinton[2] and Bowers[1] mention the absence of sacral

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concavity and the straightness of the rectum as additional etiologic factors in children.

The condition occurs with relative frequency in infants and children, although it may develop at any age. In children the first symptom is the appearance of a reddish mass which protrudes from the rectum coincident with crying or straining, or after defecation. It is frequently mistaken for hemorrhoids. Discomfort and bleeding may occur; complete or partial strangulation rarely develops. The rugae of the adult mucosa are absent and differentiation from protruding polyps depends upon the demonstration of the pedunculated attachment in the latter.

The treatment in children is largely non-surgical and includes regulation of the bowel habits by diet, elimination of laxatives, correction of malnutrition if present, and the maintenance of reduction between defecations for several weeks by strapping the buttocks together or by the elevation of the legs to a vertical position. Rarely, when the condition resists such measures, the simpler surgical procedures applicable to adults become necessary.

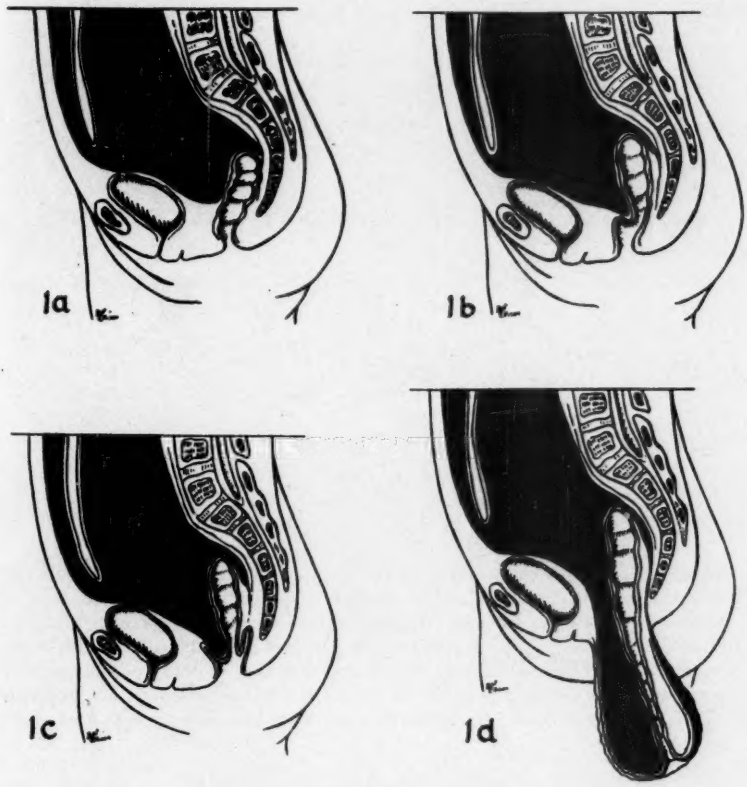
In adults, mucosal prolapse occurs more often in the aged; in its early stage it appears as an incomplete ring of mucosa which remains protruding after defecation (fig. 2a). Following constipation, diarrhea, increased intra-abdominal pressure or straining, an increasingly large mass of mucosa protrudes. This requires manual reduction after defecation and maintenance in reduction may become difficult (fig. 2b). Discomfort, pain and bleeding are frequent and strangulation is more apt to occur in the aged and poorly nourished individuals.

In mucosal prolapse the length of the prolapse is usually less than its transverse diameter, the rectal sulcus is not obliterated, the prolapsed mucosa is thinner than the entire rectal wall which presents in the complete prolapse and the rugae extend longitudinally rather than concentrically, as in the complete type.

In adults surgical treatment is indicated. It must be emphasized that this is a local condition only: intra-abdominal procedures and plastic operations on the perineal floor are not only unnecessary, but unsatisfactory. There are three useful types of procedures: (1) Circular excision of the mucosa surgically or by constricting bands over a tube. Stricture and hemorrhage are frequent complications of this procedure and it is only indicated in the presence of strangulation. Frequent examination and dilatation must be carried out for some time after such an operation. (2) Subcutaneous injections of sclerosing solutions or linear cauterizations; either gives satisfactory results. (3) Vertical excision of strips of mucosa

with undermining of the intervening mucosal segments. This is the method of choice (fig. 3).

Because of the frequency of this condition only one case history



Figs. 1. Development of complete rectal prolapse as described by Moschcowitz.

(a) Relations between the cul-de-sac and the rectum before development of hernia.

(b) Beginning of the prolapse: the apex of the cul-de-sac pushed into the anterior rectal wall.

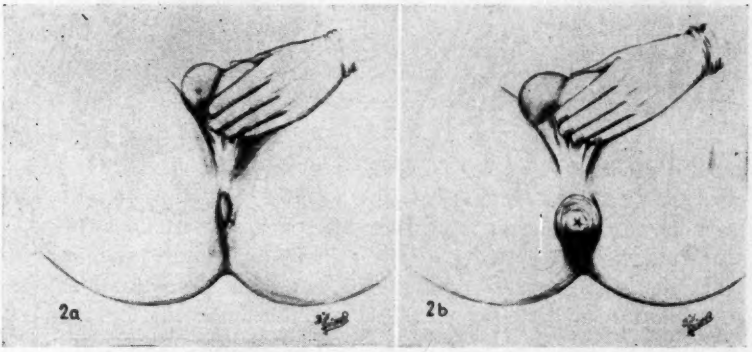
(c) The other rectal walls fold inward and follow toward the sphincter; this also represents "high prolapse" or intussesception of the recto-sigmoid.

(d) Fully developed prolapse. Note the hernial sac in the anterior wall of the prolapse.

is briefly presented.

CASE 1. A man, aged 33 years, had gradually increasing rectal protrusion of 4 years' duration. The protrusion occurred with defecation and occa-

sionally spontaneously. It was quite painful and had required manual reduction, once by a physician. He gave a history of chronic constipation of moderate degree. Hemorrhoidectomy had been done without relief. When the patient was examined the protrusion had been present 24 hours; he had not been able to reduce the mass and marked pain had developed.



Figs. 2.

(a) Beginning prolapse of the rectal mucosa.
(b) Severe prolapse of the rectal mucosa (Case 1).

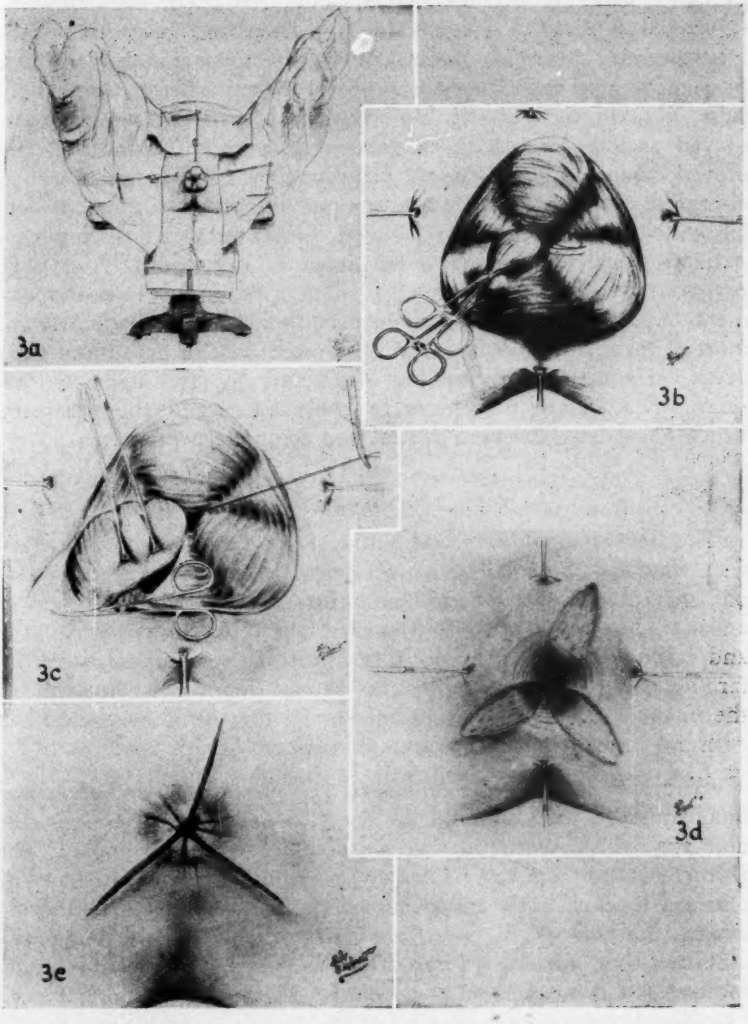
Examination revealed prolapse of the rectal mucosa the size of a baseball. There was extensive edema and bluish-red discoloration. Reduction was accomplished with some difficulty and maintained for several days with a pressure pad. The anal sphincter was relaxed, the peri-anal tissues were thickened. On July 19, 1943, under spinal anesthesia, resection of longitudinal strips of mucosa and adjacent skin was carried out. He was asymptomatic and returned to work on the eleventh day; after 14 months he still remained entirely well.

II. Complete Rectal Prolapse

Complete prolapse, or hernia, is a protrusion of the entire rectum and part of the recto-sigmoid through the anal orifice. It differs etiologically and anatomically from incomplete prolapse; hence the incomplete type does not develop into the complete prolapse, although the two types may co-exist.

Literature

The subject of complete rectal prolapse has received much attention in the literature. In 1912 Moschcowitz[3] made an outstanding contribution when he showed that the condition is not truly a prolapse of the rectum, but a sliding hernia. His work has been confirmed and accepted by the majority of subsequent investigators.



Figs. 3. Repair of prolapse of the rectal mucosa.

(a) Exposure with Allis forceps and elastic bands for retraction.

(b) A segment of mucosa is grasped with Allis forceps and ligated high up with 00 chromic catgut.

(c) Segment of mucosa excised down to sphincter muscle and extended externally to include triangle of perianal skin.

(d) The three large segments of mucosa and skin have been excised.

(e) Traction removed preparatory to insertion of stuffed Penrose drain in rectum and pressure pads externally.

Moschcowitz reported 8 patients operated upon, with excellent results in 7 after 4 months to 5 years; there was one death from uremia. Swinton[2] of the Lahey Clinic reported 7 patients, 4 of them treated with the Moschcowitz operation with excellent results. Rankin and Priestly[4] reported the Moschcowitz technic employed on 9 patients with excellent results in 7. Pemberton[5] employed colopexy on 6 patients with excellent results, but the postoperative period of observation was too short for him to draw definite conclusions in regard to its value. Other authors have reported patients treated by various other types of operations,[6-15] or have reviewed the subject at length.[16, 17] The injection methods of treatment have received considerable attention.[18-22] An evaluation of most of these other operative procedures is difficult because of their great variety, the small number of patients operated upon, the absence of adequate postoperative observation and the confusion which has existed between partial and complete prolapse.

Etiology

The development of rectal prolapse as described by Moschcowitz was that the small intestines push the apex of the cul-de-sac and the anterior wall of the rectum into the rectal lumen (figs. 1a and b). The other walls fold inward and follow toward (fig. 1c) and through the sphincter to appear externally as a gradually enlarging mass (fig. 1d). This mass should then consist not only of the inverted rectum, but also anteriorly of a peritoneal sac filled with intestines. This has repeatedly been observed.

Abnormal depth of the cul-de-sac, and redundancy and abnormal mobility of the rectosigmoid are constant findings in these patients and probably essential to the production of the prolapse. The former results from the failure of the cul-de-sac to ascend from its perineal level of early embryonic life, and the redundancy and abnormal mobility of the bowel are also probably largely congenital, although they may be increased by the traction of the developing hernia. All abdominal hernias must emerge either through a perivascular or perivisceral opening in the transversalis fascia. In this instance the perivisceral opening of the rectum provides its point of emergence, and congenital defects, or acquired stress, or both, may be supposed to provide the essential weakness of this opening. Such acquired stresses include increased intra-abdominal pressure due to lifting, parturition, ascites, straining with constipation, diarrhea, coughing or urethral obstruction, or atrophy of the tissues incident to advancing age, malnutrition and wasting disease. The incidence in the insane is apparently high.[1, 9, 23]

Moschcowitz and others have observed that pressure by the finger anteriorly inside the rectum will prevent prolapse even with severe straining, but if pressure is exerted in any other direction and not anteriorly, the prolapse promptly recurs with straining. Pressure on the apex of the cul-de-sac toward the anterior wall of the rectum during laparotomy reproduces the hernia. These findings are all consistent with and confirm this conception of the etiology of the condition. The role of an incontinent sphincter is probably negligible because rectal prolapse does not occur when incontinent sphincter develops and a continent sphincter usually results after correction of a rectal prolapse without treatment of the sphincter itself.

Symptoms and Findings

Complete prolapse of the rectum occurs more often in women than in men, the ratio being about two to one. The age of onset is highest early in childhood and in later decades of life, suggesting both a congenital factor in the former and a relationship to stress in the latter.

The cardinal symptom is a mass which protrudes from the rectum. The onset may be gradual, and progressive enlargement may occur for many years and then cease. Early, the protrusion occurs only with straining or defecation. After months or years it attains a maximum size and as the sphincters and surrounding structures become dilated, it appears when walking or coughing and later spontaneously. Manual reduction and later pressure pads become necessary to maintain reduction. Less often the mass appears fully developed after severe strain. Other symptoms are: a mucous discharge, a feeling of discomfort and fullness, incontinence of feces and gas. Less frequently there is pain, bleeding and backache.

Examination reveals the large inverted cone-shaped mass protruding from the rectum. It is covered by mucosa which may be normal or markedly inflamed and thickened. The rugae are circular. The bowel opening is slightly posterior to its apex (fig. 4) because it is the anterior wall of the rectum which is most relaxed and redundant. The presence of intestines in the anterior portion of the mass may produce a tympanitic note on percussion, and gurgling on manual reduction was noted by Moschcowitz. The perianal sulcus may be present or it may be obliterated when the lowermost segment of the rectum is involved.

The patient who gives a history of such a protruding mass may present no external evidence of it because of its reduction at the time of examination. When an enema is given, however, or the

patient strains, the mass becomes apparent. The sphincter may have a fair tone or be so completely relaxed that voluntary contraction cannot be elicited and it often readily admits several fingers or the entire fist. Rectocele, cystocele or prolapse of the vagina occasionally co-exist.

The differentiation from the incomplete prolapse is based on five points: (1) The greatly increased size and thickness of the complete prolapse; (2) its length is usually greater than the diameter of its base (it is about 6 inches or more when fully developed); (3) the opening in the gut is posteriorly placed; (4) the rugae are transverse instead of vertical as in partial prolapse; (5) there is tympany on percussion in the presence of intestine in the associated hernia.

Treatment

The treatment of complete prolapse of the rectum is surgical unless contraindicated by the general condition of the patient.

The surgical procedures that have been suggested are so numerous that it would be impossible even to enumerate all of them in a paper of this scope. This is indeed unfortunate because it makes the selection of a suitable operation most difficult. We will attempt to analyze them in groups and point out the indications for those few which appear rational and useful.

A: Perineal Operations

Group I: Procedures Designed to Prevent Prolapse by the Formation of Adhesions to the Rectum.

A: Injection methods with paraffin, alcohol, quinine and urea hydrochloride, phenol and other irritants.

B: Cauterization of the mucosa in vertical strips.

C: Longitudinal resection of the mucosal strips with undermining of the intervening mucosa.

Comment: The groups A, B and C do not correct any of the anatomic abnormalities present in the complete prolapse; they have to be repeated frequently or fail altogether, are not without risk and are, in the opinion of most writers, only palliative. They are applicable and curative in mucosal prolapse only. Attachment of the rectum to the anterior sacrum is an unsound procedure because it fails entirely to correct the abnormal mobility of the anterior rectal wall which is responsible for the occurrence of the prolapse.

Group II: Procedures to Narrow the Anal Outlet.

A. Circular cauterization of the rectal mucosa with the actual cautery or with corrosives.

B. Resection of a segment of the posterior wall of the rectum and suture of the edges.

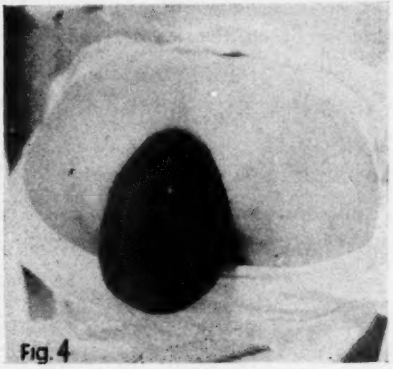


Fig. 4. Complete rectal prolapse, severe (Case 3). Note posterior position of the bowel opening.

C. Encirclement of the anal outlet with silver wire.

D. Removal of elliptical section, reefing of the submucosa and suture, or removal of cuff of mucosa from the outer cylinder with reefing and suture of mucosa to skin.

E. Twisting of the rectum.

COMMENT: These procedures do not correct the primary defect responsible for the prolapse. They deal only with the most external barrier to the complete prolapse which is not necessarily weak as shown by the failure of prolapse to develop when the sphincter is totally incontinent or even destroyed. The sphincter will regain its normal function spontaneously after correction of the prolapse unless it has been previously destroyed. Removal of the mucosa and reefing of the submucosa have the added effect of shortening the extruded segment of bowel and utilizing it to strengthen the peri-rectal support. Its ability to attain permanent cure is open to question.

Group III: Amputation of the Bowel from Below.

A. Insertion of a tube or cylinder and amputation by constriction with a suture, wire or elastic.

B. Complete surgical amputation and suture.

COMMENT: These methods, especially group A, are applicable when strangulation, gangrene or extensive infection are present to an extent that prohibits the return of the bowel to its normal habitat. The procedure is not infrequently complicated by hemorrhage and followed by stricture, as well as by recurrences.

Group IV: Repair or Strengthening of the Perineal Floor from Below.

A: Repair of the sphincter, approximation and suture of the levator muscle.

B: Muscle plastics utilizing the glutei muscle.

C: Closure of the cul-de-sac through an incision anterior to the rectum and repair by suture and sometimes reefing of the levator. (Napalkow.)

COMMENT: All of these methods are difficult and repair only the perineal floor; they again neglect the etiologic abnormality. Moschcowitz commends the Napalkow procedure to a certain extent but objects to it because there is no true hernial sac to extirpate and because of its difficulty when performed from below.

B: INTRA-ABDOMINAL OPERATIONS

Group I: Colopexies: These Have Utilized One or Several of the Following Procedures:

A: Re-attachment of the sigmoid or recto-sigmoid at a higher level or at a different angle.

B: Suture of the sigmoid or recto-sigmoid to the abdominal wall, usually anteriorly.

C: Obstructive resection of the sigmoid with colostomy which is subsequently closed.

COMMENT: These operations are not altogether without risk and may be difficult, depending upon whether or not the recto-sigmoid is resected. They support the colon but do not primarily obliterate the sac or block the point of exit of the hernia. Moschcowitz stated that there is almost invariably recurrence after a sufficient period of observation.

Group II: Repair of the Perineum from Intra-Abdominal Approach

A: By denuding the perineal floor and approximation and suture

of the levators and fascia of the floor to force the rectum backward and narrow the outlet (Roscoe Graham[10]).

COMMENT: These operations rely only on the narrowing of the rectal opening in the perineum and fail to deal with the sac or protect the point of weakness in the anterior rectal wall.

Group III: Elevation of the Floor of the Cul-de-Sac.

A. By obliteration of the cul-de-sac with a series of purse-string sutures at increasingly higher levels and, in addition, in older women, attachment of the uterus to the abdominal wall (Moschcowitz).

B. Reformation of the pelvic floor at a higher level by suture of the peritoneum of the cul-de-sac over a framework built of fascial strips (Mayo[25]).

COMMENT: The Moschcowitz repair has seemed to give the most satisfactory results over the longest period of time and has the advantage of extreme simplicity of performance and minimum of risk. It accomplishes obliteration of the hernial sac, elevation of the pelvic floor to such a height that the prolapsing small intestine cannot reach the area of lowered resistance in the anterior rectal wall, it straightens and supports the sigmoid and recto-sigmoid and forms a large tampon which effectively blocks the hernial opening. Moschcowitz, Rankin, Swinton and others report series of patients treated by this method with uniformly good results after years of observation.

Its usefulness is admitted even by those who advocate colopexies when they have utilized it as an additional safeguard in many of their cases. It is not improbable that the effectiveness of colopexy may depend to some extent upon the degree to which the cul-de-sac coincidentally is obliterated.

The Mayo procedure is well devised but seems unjustifiably complex, when raising of the pelvic floor is so much more easily and just as effectively accomplished by the Moschcowitz procedure.

CASE REPORTS

Two patients with complete prolapse of the rectum are reported, both treated by the Moschcowitz procedure.

CASE 2: A woman, aged 55 years, para XI, examined in October, 1934, had developed "hemorrhoids" after her first pregnancy 30 years previously. With the second and third pregnancies they enlarged until manual reduction became

necessary and bleeding frequently occurred. Hemorrhoidectomy, 18 and again 12 years previously, had resulted in less bleeding and only very temporary improvement. Then the protrusion increased until a mass larger than a baseball presented after coughing, walking or defecation: it was manually, but not spontaneously, reducible. Incontinence of gas and feces progressed for 18 years until complete. Constipation, present for 30 years, required moderate catharsis.

On examination the perianal skin was thickened and leathery; scarring was present, anteriorly and posteriorly. Marked relaxation of the sphincter ani resulted in total loss of control. Straining or coughing caused protrusion of the rectum to produce a long, thick-walled mass, larger than the size of a fist. The rugae were circular, the bowel opening posterior. Complete prolapse of the rectum was diagnosed.

First-stage Operation, Oct. 31, 1934: With prolapse reduced, a transverse incision was made midway between the rectum and the coccyx and the rectum exposed up to the peritoneal reflection. Four silkworm sutures were placed transversely across the rectum posteriorly. The ends were brought out through the skin on either side of the sacrum at a slightly higher level, and tied over a piece of gauze, elevating and fixing the rectum up into the hollow of the sacrum. The incision was closed and a purse-string suture of kangaroo tendon was placed around the rectum at about the level of the sphincters and tied subcutaneously, reducing the rectal lumen to admit only the tip of the finger.

The patient was kept in bed, sutures removed on the 14th day. The wound healed, there was no prolapse, and the sphincter control was about 80 per cent efficient.

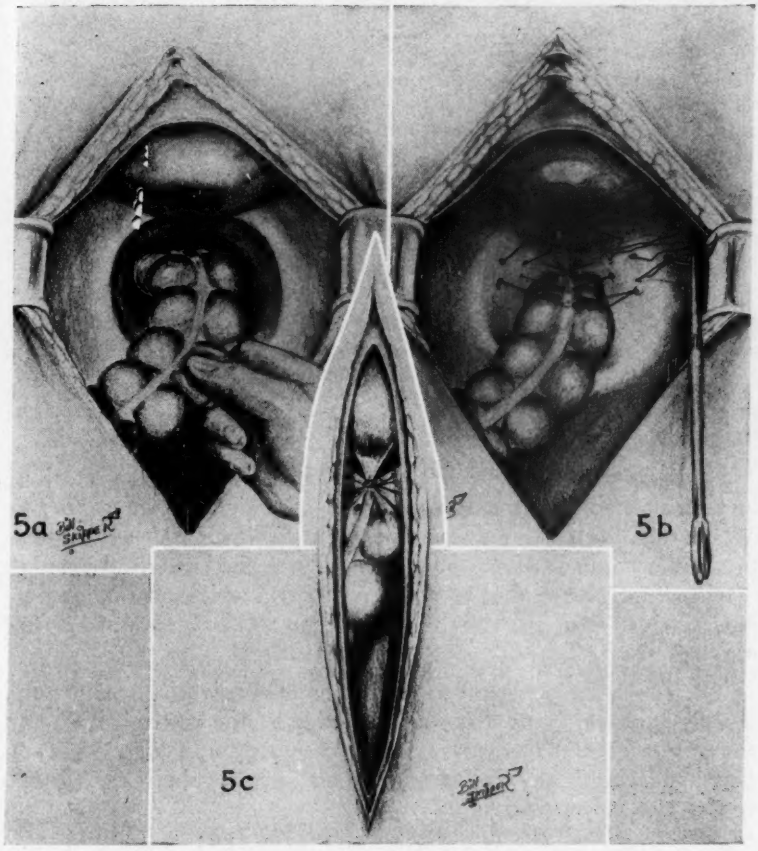
Second-stage Operation: On the 24th day, under ether anesthesia, a low midline incision was made. The cul-de-sac was very deep and the uterus was atrophic. The recto-sigmoid was markedly redundant. The sigmoid was pulled up under tension and the cul-de-sac was obliterated by the Moschcowitz procedure (fig. 6). The fundus of the uterus was excoriated and sutured into the abdominal wall. Convalescence was uneventful and recovery complete.

On May 13, 1936, there was slight prolapse of the mucosa about 1 cm. in diameter on either side. At operation, under local anesthesia, a vertical strip of mucosa was excised anteriorly and the sphincter tightened again with a kangaroo tendon and a dressed #18 catheter placed in the rectum.

When the patient was last contacted in 1946, there were no further symptoms.

COMMENT: The first-stage operation carried out in this patient is not considered essential and would not again be utilized.

CASE 3: A woman, aged 33 years, was admitted to the Birmingham Baptist Hospital on the service of Dr. J. P. Motley on May 24, 1935. In 1920, at parturition, there were lacerations, and hemorrhoids appeared. In 1923 she had been given massive doses of calomel during an attack of measles. Tenderness developed in the rectum and two months later there was bleeding with bowel movement. Hemorrhoidectomy and perineorrhaphy in 1924 resulted in relief of symptoms for six months, when the protrusion recurred and gradually enlarged manual reduction became necessary after defecation. Hemorrhoidectomy in 1928 was without improvement.



Figs. 5. Repair of complete prolapse of the rectum in male (Moschcowitz technic).

(5a) Low midline incision with the small intestine packed off; the sigmoid is grasped and pulled upward to reduce the prolapse.

(5b) Series of purse-string sutures include the sigmoid and the lateral and anterior pelvic walls.

(5c) Sutures tied completely obliterating the cul-de-sac preparatory to closure of the abdominal incision.

The patient was moderately underweight. The pelvic floor was not relaxed and the uterus was in good position and the adnexa appeared normal. The rectal sphincter was markedly relaxed and easily admitted three fingers. Straining or bowel movement resulted in complete eversion of the rectum to form a protruding cylindrical mass approximately one foot in length and four inches in diameter (fig. 4) at its base. The rugae were circular, the bowel opening posterior. A diagnosis was made of complete prolapse of the rectum and recto-sigmoid.

Operation May 25, 1935: A low midline incision was made. The cul-de-sac

appeared deeper than normal. The sigmoid was moderately redundant and the wall of the bowel in the lower recto-sigmoid and rectum was markedly thickened and hypertrophied to several times its normal size.

The prolapsed rectum and the recto-sigmoid were completely reduced by traction on the bowel from above, and the cul-de-sac obliterated by the Moschcowitz technic (fig. 6). Bilateral salpingectomy and Gilliam suspension of the uterus were performed. The convalescence was uneventful. In 1946 no protrusion could be induced and there was normal rectal control. A fibromyoma of the uterus had regressed after radiotherapy.

III. High Prolapse of the Rectum

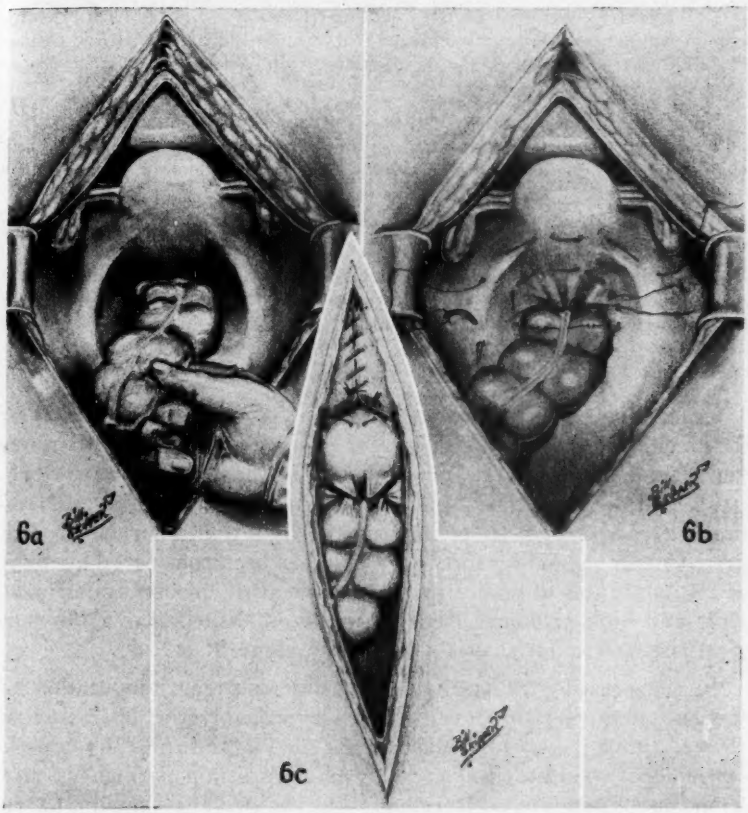
High, blind, or third degree, prolapse of the rectum is not a true prolapse, but an intussusception of the recto-sigmoid into the rectum which does not present externally. The condition has received very scant consideration in the literature.[26] It probably occurs much more often than it is recognized because the diagnosis is not obvious unless specific search is made for it.

Symptomatically it may be acute or chronic and is characterized by an ineffectual desire to defecate and often the passage of mucous and blood, intermittent cramping pain in the lower abdomen, distention and occasionally vomiting. Lying flat or the knee-chest position often relieves the pain. On digital or proctoscopic examination during the episode of pain the presenting tip of the telescoped bowel may be identified.

The etiology is similar to that of complete prolapse of the rectum, the invagination occurring at the junction of the recto-sigmoid and the rectum. It is probable that many of these cases progress to complete prolapse. The treatment is surgical, preferably by the Moschcowitz type of operation.

Case 4 illustrates this condition: it was treated surgically by the Pemberton type of colopexy. Although no recurrence of the intussusception could be demonstrated, symptoms persist to a milder degree than before operation and it is believed that the Moschcowitz type of operation would have been preferable.

Case 4: A nurse, aged 46 years, complained of low abdominal cramps, distention and vomiting. At the age of 10 she had measles, with severe coughing. Following this the pain started in the low abdomen, around the navel, then to the back and down toward the legs. It came in attacks and the pain recurred every few minutes, often associated with distention and vomiting which frequently awoke her at night. The condition gradually became worse, especially after pyelitis and cystitis, and amebic dysentery in 1938 and 1939. The pain was aggravated by lifting or straining at stool, at times by diarrhea or constipation or eating large meals. Occasionally she could feel a mass in the left lower abdomen and again she had a persistent desire to defecate without relief. These conditions were usually relieved when she assumed the



Figs. 6: Repair of complete prolapse of the rectum in the female.

(6a) Low midline incision with the small intestines packed off. The sigmoid is grasped and pulled upward to reduce the prolapse.

(6b) A series of purse-string sutures include the sigmoid, the utero-sacral ligaments, the posterior aspect of the vagina and lower uterus, and the lateral walls of the pelvis.

(6c) Sutures tied and the uterus sutured into the lower portion of the abdominal wound preparatory to its closure.

knee-chest position. Recently there were alternating diarrhea and constipation and sometimes the passage of bloody mucus and a little gas. Enemas were often ineffective.

In 1939 she had had hemorrhoidectomy and in 1941 an operation for removal of an abdominal tumor which was said to be causing obstruction. All were without relief.

She was moderately well-nourished, very introspective. Digital examination of the rectum was negative except after bowel movement or straining, when a large boggy mass could be felt presenting about two inches inside the rectum. It was doughnut-shaped and the finger could be passed all around it

or into a central opening. In the knee-chest position it disappeared. Proctoscopic examination was negative except that upon withdrawal with the patient straining, any point high in the recto-sigmoid followed the proctoscope downward for an abnormal distance.

Intussusception of the recto-sigmoid was diagnosed and on Sept. 23, 1944, a Pelberton colopexy was performed. There were numerous adhesions around the sigmoid and the sigmoid was abnormally long and loosely attached by a long mensentery. Convalescence was uneventful.

Six months later there had been some difficulty with constipation. Pain had been present much less often and less severe and she had gained weight and felt much better. Nervousness and menopause symptoms were severe. On examination the rectal wall appeared redundant. Prolapsing bowel could not be demonstrated even with severe straining.

Summary and Discussion

Cases illustrating the various types of rectal prolapse and their treatment are presented. The great number of surgical procedures that have been suggested, particularly for the complete rectal prolapse, makes the selection of the most appropriate difficult. However, since Moschcowitz considered the condition a hernia, his suggested cul-de-sac obliteration operation has been considered by many as the operation of choice in the treatment of uncomplicated complete and high prolapse. It is easily accomplished, has a low surgical risk and accomplishes a high percentage of cures.

In the presence of strangulation and gangrene, amputation of the rectum by ligation over a tube is probably the operation of choice, although stricture must be guarded against and the Moschcowitz operation may be necessary later to attain permanent cure. Coincident cystocele, rectocele or uterine prolapse should be repaired shortly before or after, or coincident with, the Moschcowitz operation, depending upon the condition of the patient. Before menopause, tubal ligation is indicated because pregnancy would favor recurrence. The persistence or appearance of a small amount of prolapse postoperatively is frequently due to a coincident mucosal prolapse and may require supplemental operation for its correction.

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BIBLIOGRAPHY

1. Bowers, F.: Treatment of Prolapse of the Rectum, Nebraska M. J. *24:* 419-423 (Nov.) 1939.
2. Swinton, N. W.: Prolapse of the Anus and Rectum, S. Clin. North America, *20:* 829-841 (June) 1940.
3. Moschcowitz, A. V.: The Pathogenisis, Anatomy, and Cure of Prolapse of the Rectum, Surg., Gynec. & Obst. *15:* 7-21 (July) 1912.
4. Rankin, F. W., and Priestly, J. T.: Prolapsé of the Rectum, Ann. Surg. 1030-1038 (Dec.) 1933.

5. Pemberton, J. deJ., and Stalker, L. K.: Surgical Treatment of Complete Rectal Prolapse, Ann. Surg. *109:* 799-808 (May) 1939.

6. Hiller, R. I.: Experience with the Rehn-Delorme Operation for Rectal Procedentia, M. Times, New York, *70:* 191-194 (June) 1942.

7. Martin, E. G.: Prolapse of the Rectum, J.A.M.A. *99:* 368-372 (July 30) 1932.

8. Smith, G. V. S.: Modification of an Old and Simple Method of Treating Rectal Prolapse, New England J. Med. *232:* 495-497 (May 3) 1945.

9. Elliason, E. L.: Rectal Prolapse. Experience with the Elastic Ligature, Ann. Surg. *105:* 199-302 (Feb.) 1937.

10. Graham, R. R.: The Operative Repair of Massive Rectal Prolapse, Ann. Surg. *115:* 1007-1014 (June) 1942.

11. Reid, M. R.: Irreducible Prolapse of the Rectum, Am. J. Surg. *20:* 359-365 (May) 1933.

12. Barrett, C. W.: Hernioplastic Operations upon the Rectum and Pelvic Floor, Am. J. Surg. *59:* 519-525 (March) 1943.

13. Daniels, E. A.: Prolapse of the Rectum, Internat. Clin. *4:* 203-217 (Dec.) 1938.

14. Chapple, H.: Prolapse of the Rectum in Women, Brit. M. J. *1:* 661-662 (May 12) 1945.

15. Murdoch, R. L.: Prolapse through the Anal Sphincters, South. M. J. *31:* 1091-1093 (Oct.) 1938.

16. Salvin, A. A.: Surgical Treatment of Rectal Prolapse, Ann. Surg. *101* 1051-1061 (April) 1935.

17. Cohn, I.: Prolapse of the Rectum, Am. J. Surg. *57:* 444-449 (Sept.) 1942.

18. MacEwen, J. A. C.: Treatment of Rectal Prolapse by Injection, Brit. M. J. 633 (April 14) 1928.

19. Morley, A. S.: Injection Treatment of Complete Rectal Prolapse, Brit. M. J. *2:* 204-206 (Aug. 4) 1934.

20. Lee, M.: Treatment of Partial Prolapse of the Rectum by Perirectal Injection of Sylnasol, Proc. Roy. Soc. Med. *32:* 1498-1500 (Sept.) 1939.

21. Fansler, W. A.: A Resume of Partial Rectal Prolapse with a Suggestion as to Treatment, Minnesota Med.: 372-375 (June) 1932.

22. Galbraith, D.: A Simple Treatment for Rectal Prolapse, M. J. Australia *2:* 620 (Nov. 14) 1931.

23. Fair, G. L.: Prolapse of the Rectum in the Insane, New York State J. Med. *38:* 641-645 (April 15) 1938.

24. McDonald, E. P.: Prolapse of the Rectum and Uterus in the Same Patient, Am. J. Surg. *15:* 47-50 (Jan.) 1932.

25. Mayo, C. W.: Complete Rectal Prolapse, West. J. Surg. *46:* 75-77 (Feb.) 1938.

26. Monsarrat, K. W.: High or Third-degree Prolapse of the Rectum, Brit. J. Surg. *14:* 89-93 (July) 1926.

RECENT DEVELOPMENTS IN SURGERY

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AMERICAN surgery may be divided into five epochs: the era up to and comprising the time of Ephraim McDowell; the Watson, Morton, Long period including the introduction of general anesthesia; the aseptic age of Pasteur and Lister; the period of the surgical giants, Halstead, Senn, Murphy and Ochsner; the clinic era of the Mayos and Crile; and finally the present epoch when improved preoperative and postoperative care, chemotherapy, spinal anesthesia and early ambulation have proved so important a role in surgery.

The clinic era witnessed the passing of the general surgeon, one who could reduce a fracture, remove a gallbladder, a cord tumor, or a kidney stone with equal dexterity. A few of these "all around" brilliant surgeons remain, but their number is decreasing as they give way to the age of specialization. Today surgery has become the most highly specialized branch of medicine, with many surgeons limiting themselves largely to the abdomen, the chest, the nervous and skeletal system, the genitourinary tract and the pelvis. No longer can any one surgeon hope to cover these many branches of surgery and achieve the same results obtained by those specializing in one or two of these fields. This is an age of specialization and a time of highly perfected teamwork in the operating room. Great as were the surgical giants of the nineties, few could have matched the brilliant surgery of such a team as the late Starr Judd and Fred Rankin. So today, teamwork has become a necessity in most operating rooms in America. Yet, speed is no longer a prime requisite because spinal and nerve block anesthesia have largely superseded the use of ether. Rather, teamwork is important in perfecting the details of surgical technic and in the preoperative and postoperative care of the patient. Certainly the greatest advance in this era of surgery has been in this field.

Through the use of chemotherapy the death rate from all types of surgery has been materially reduced in the past five years. At the Atlanta meeting of this Congress, I discussed this phase of surgery and today I will only summarize my experience since that time.

From the Jackson Clinic, Madison, Wisc.

Read before the fourteenth annual Postgraduate Surgical Assembly of The Southeastern Surgical Congress, Memphis, March 11, 1946.

At the Jackson Clinic we have used sulfathiazole in over 4,000 surgical cases with the most gratifying results. It is used in the closure of all incisions, is freely employed within the abdominal cavity, and has been an important factor in reducing infection. At Atlanta I had insufficient data to advocate closure of most abdominal wounds without drainage in the presence of a ruptured appendix and peritonitis. Experience has led me to close all such cases without drainage after the introduction of 8 to 12 Gm. of sulfathiazole in saline into the peritoneal cavity. The elimination of profuse purulent discharges, of painful and tedious dressings, of complications such as obstruction and abscess, and of the long, expensive periods of hospitalization has been a blessing to the patient, nurses and surgical staff. A few progressive surgeons such as Alton Ochsner had the courage to close such cases without drainage before the advent of the sulfonamides, but most American surgeons employed drainage in perforated appendicitis and still do.

Only the initial local dose of sulfathiazole has been used at our clinic, and as a consequence, we have seen no complications of any serious nature, such as anuria. The only change in our treatment has been the giving of penicillin, 30,000 units either intravenously or subcutaneously, every 3 hours for a few days. It is difficult to evaluate how much benefit the patient has derived from this because satisfactory results were obtained before the use of penicillin.

Despite advances in surgery, improvements in transportation, and erection of modern hospitals, the mortality rate from appendicitis in the United States had increased from 9.7 per 100,000 population in 1900 to 12.8 in 1936. During the year 1939 two persons died every hour from appendicitis in this country. In 1941 the use of sulfanilamide within the peritoneal cavity for perforated appendicitis was first advocated by the Roosevelt Hospital group in New York City, and since that time the death rate has steadily decreased. Nearly 17,000 persons died annually prior to the use of sulfonamides; now less than 9,000 succumb. A communication received March 4, 1946, from Dr. H. L. Dunn, Chief, Vital Statistics Division, U. S. Bureau of Census, stated that the latest available figure was for 1943, and showed 8,108 deaths from appendicitis in this country. Probably the use of penicillin will result in a further decrease in these figures.

Use of the sulfa drugs and penicillin has likewise reduced the mortality rate in other types of surgery such as in perforations of the gastrointestinal tract, traumatic lesions, ulcers, compound fractures, carcinoma of the large bowel and chest surgery. It has been

possible to do more one-stage large bowel resections with less risk, and a great saving in hospitalization to the patient has resulted.

In recent years more attention has been paid to the careful preoperative preparation of all types of malignant cases of the gastrointestinal tract. The wasted tissues have been strengthened with vitamin therapy, with more generous use of blood transfusions and amino acid preparations so that postoperative eviseration of the wound has become a rare complication. In addition, the use of the sulfonamides and penicillin has reduced and virtually eliminated other postoperative complications such as pleurisy, pneumonia, and empyema.

Thiouracil, another valuable chemical aid, has lessened the problem of the thyroid surgeon.[1] After three years of clinical investigations in 5,751 cases this drug is now released to the medical profession; yet out of 160 cases 21 deaths from agranulocytosis have been reported. Nevertheless, it has proved the means of saving many patients from developing advanced thyrotoxicosis. It has been found beneficial in nearly 84 per cent of over 2,000 cases treated.

My experience in a small series of 50 patients treated during the past three years has led me to the following conclusions: thiouracil will not supplant thyroidectomy in the treatment of multiple toxic adenomata, but in preoperative preparation it is more effective than Lugol's solution. Iodine benefits 32 per cent of this type, but 80 per cent improve on thiouracil, and it is especially valuable if considerable damage to the cardiovascular system has occurred and if the surgical risk is high.[2] In these cases a one-stage rather than a two-stage thyroidectomy can be performed with comparatively little danger. This has been a great advance in the field of thyroid surgery.

In the "iodine-fast" exophthalmic goiter, a bad surgical risk, where iodine has been used so long it has lost its beneficial effect, thiouracil has proved effective as a means of resting the patient from iodine while preventing him from losing ground. In late pregnancy with exophthalmic goiter it is possible to carry the patient through term on thiouracil and then after delivery change to iodine and perform a thyroidectomy. I have three cases of recurrent exophthalmic goiter treated with thiouracil now symptom-free six months or more following withdrawal of the drug.

It takes years and a large series of cases properly to evaluate such a drug, so no one is yet prepared to say how many and what type of cases of exophthalmic goiter might be cured by this method. So far, there has been only one primary case in my experience that

has become symptom-free and in which the goiter has disappeared. Possibly if I had given my patient's larger doses of thiouracil and continued treatment for longer periods, more cures might have occurred, but I did not feel justified in subjecting them to this risk or in delaying cure so many months when the latter could so quickly and safely be obtained by thyroidectomy. Thiouracil has proved of valuable assistance to the surgeon in the treatment of hyperthyroidism, but it is to be hoped that a less toxic drug may be discovered to replace it. My experience indicates that propythiouracil may fulfill that hope.

In December, 1944, I was invited to speak to the St. Paul Surgical Society on "Recent Advances in Surgery." Mention was made of some developments in surgical technic, but special stress was laid upon changing ideas in our postoperative care. Among other things, I advocated getting nearly all surgical patients out of bed the day after operation, curtailing the use of intravenous glucose and saline, substituting early feeding, reducing and eliminating enemas when possible, and using a simple sulfathiazole Mediplast dressing in place of the large, bulky gauze ones, abdominal pads and binders. At that time there might have been reason to question the rationale of some of these ideas, but nearly two years' experience has convinced me of the merits of these changes in the usually accepted methods of postoperative care.

Early ambulation of operative cases has proved an important factor in decreasing complications and in lowering the mortality of my surgical cases. American surgeons cannot claim credit for suggesting early ambulation following operation, as both the French and German surgeons advocated this many years ago.[3] In Germany, for instance, Mermingas operated upon his patients for appendicitis under local anesthesia and then had them walk immediately to their rooms. In 1899 Ries[4] first advocated early ambulation to American surgeons; but it was not until 1941 that interest in this subject really awakened when Leithauser and Bergo[5] presented a series of 484 cases using early postoperative walking. At that time I was getting most of my patients out of bed on the fourth postoperative day. For the past eighteen months I have had the majority of patients out of bed in twenty-four hours, taking a few steps the next day, and sitting in a chair the following day. During this period there have been no complications that could be attributed to early rising. Intestinal resections, cholecystectomies, appendectomies for perforated appendices, herniotomies, hysterectomies and thyroidectomies have all been treated alike.

To walk into a room and see a patient sitting reading a magazine

two or three days after undergoing major surgery is indeed a revelation that would have startled most of us not so long ago. Like so many of our ideas in surgery that have been handed down on a basis of tradition rather than fact, I believe it was a mistake to keep these patients in bed for one, two or even three weeks. With early rising the circulation is speeded up; hence the biggest factor in causing phlebitis and embolism is eliminated. The wound heals much more quickly because of the improved circulation to the damaged structures. Also, none of the pulmonary complications previously occurring in 3 or 4 per cent of the cases have been observed in my series in the past year and a half. The recumbent position not only results in congestion, stasis and edema of the lungs, but it favors the accumulation of excessive mucous secretions. Early rising favors the return of the cough reflex which serves to get rid of these secretions and reduces the splinting of the diaphragm, allowing greater expansion of the lungs and lessening the tendency to pulmonary congestion and collapse.

The recumbent position further adds to the patient's discomfort by slowing down peristalsis, so that any attempt at eating is usually followed by gas pains. If the patient is up and about, peristalsis is stimulated along with the increased blood flow and food can more easily be tolerated; therefore, there is a tendency to a normal bowel movement. Enemas, intravenous therapy, and duodenal suction may consequently become unnecessary. Frequently enemas are not properly given and by further increasing distention only add to the patient's distress and increase the tendency to ileus. Early rising reduces the degree of postoperative gastrointestinal atony, lessens the tendency to nausea, vomiting, constipation, and hemorrhoids. Patients are gratified to be able to use the toilet rather than the bedpan.

The remarkable recoveries seen as a result of early rising after major surgical operations are beginning to make me feel that neither the kind of suture material nor the type of incision is of such great importance as we have tried to argue ourselves into believing. I have always been content to use fine chromic sutures in closing abdominal incisions; but because I feared a possible wound separation from getting the patient out of bed the day after operation, I have been using in addition tantulum wire no. 30 in interrupted figure of eight sutures in closing abdominal incisions. However, in herniotomies, appendectomies, vaginal hysterectomies and colporrhaphies no unfavorable results have occurred after using only fine chromic sutures. Possibly, then, silk, cotton, nylon and even wire sutures are not the answer, but early ambulation and fine chromic catgut the real solution.

Early rising cannot be considered a routine procedure and cure-all, but in the majority of surgical cases it may be used safely. There will always be certain contraindications such as shock, severe anemia, extreme debility, serious heart lesions, high fever, the possibility of hemorrhage, severe abdominal distention and rigidity. Duodenal suction and the use of intravenous fluids may be required for a day or two in serious cases, but with the exception of the sulfonamides, early ambulation has been the most important factor in improving my surgical results in the past five years.

One could discuss many other recent notable advances in surgery that have occurred, for example: refrigeration anesthesia; operation on the heart for pulmonary stenosis; treatment of carcinoma of the esophagus, the lung and the pancreas; and developments in surgery of the sympathetic system. Yet, like the explorer, the surgeon is finding that his area of exploration is becoming more and more restricted and that now his great opportunity lies in lowering the mortality of surgery through improved methods of anesthesia, the perfection of operative skill and teamwork, and the utilization of chemotherapy, early ambulation, and other developments in preoperative and postoperative care.

REFERENCES

1. Jackson, A. S., and Freeman, H. E.: The Effect of Iodine in Adenomatous Goiter, with Especial Reference to Toxic Adenomas, J. A. M. A. *106:* 1261-1263 (Apr. 11) 1936.
2. Jackson, A. S.: The Use of Thiouracil in Hyperthyroidism, Am. J. Surg. *67:* 467-478 (March) 1945.
3. Mermingas, K.: Die Appendektomie ohne folgende Muskelnaht, Zentralbl. f. Chir. *60:* 553 (Mar. 11) 1933.
4. Ries, E.: Some Radical Changes in After-Treatment of Celiotomy Cases, J. A. M. A. *33:* 454-456 (Aug. 19) 1899.
5. Leithauser, D. J., and Bergo, H. L.: Early Rising and Ambulatory Activity after Operation; Means of Preventing Complications, Arch. Surg. *42:* 1086-1093 (June) 1941.

INDICATIONS FOR SPLENECTOMY

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IN 1933 in Atlanta, Georgia, I presented before this Congress a paper dealing with splenectomy in which I attempted to summarize the indications for splenectomy in the light of knowledge that existed at the time.[1] During the thirteen years that have passed since that time, my own experience in the problems of splenomegaly, diseases of the blood-forming organs, and splenectomy, has been considerably augmented, to the extent that well over eighty of my patients have now been splenectomized for a variety of conditions. This has permitted further opportunity more properly to evaluate the results in many of these patients. Also, during this time, new facts have come to light concerning the function of the spleen, with particular respect to its phagocytic and hemolytic functions, so that the indications for splenectomy are now considerably increased over what they were several years ago, especially in view of the fact that new syndromes have been described for which splenectomy appears to be beneficial or curative.

It is the purpose of this paper again to review this subject, with particular reference to the newer diseases and clinical syndromes that apparently are benefited or cured by splenectomy. As Dameshek[2] has so well emphasized in a recent editorial in the new journal of hematology, "Blood," "These abnormal states of the spleen, particularly splenic neutropenia and pancytopenia, and the great importance of splenectomy as a life-saving measure in these cases, are just beginning to be appreciated."

At the time I presented my first paper on this subject to this organization, I listed the indications for splenectomy as follows:

1. The spleen of trauma and hemorrhage.
2. Primary tumors.
3. Essential thrombocytopenic purpura.
4. Congenital hemolytic icterus.
5. Banti's syndrome (in some cases).
6. Gaucher's disease and similar conditions.

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In listing the indications for splenectomy today, I would consider the following conditions and diseases as being true indications for removal of the spleen:

1. Spleen of trauma and hemorrhage.
2. Primary tumors of the spleen.
3. Congenital hemolytic icterus.
4. Essential thrombocytopenic purpura.
5. Gaucher's disease.
6. Banti's syndrome (in some cases).
7. Primary splenic neutropenia.
8. Primary splenic panhematocytopenia.

It will be noted that the essential difference in the two lists involves the inclusion of the last two syndromes; namely, primary splenic neutropenia and panhematocytopenia.

The Anatomy and Physiology of the Spleen

The spleen is a much larger organ under normal conditions than is generally appreciated. At the time of its removal, at the operating table or at autopsy, it is usually seen to be an organ that weighs about 125 to 150 Gm., and is about one-half as large as one's closed fist. However, under normal conditions during life, the organ is usually three to five times larger than it appears to be at the operating table or at autopsy. This is because the spleen is a large vascular lake, or reservoir, designed for the sluggish passage of blood. Its outer wall is a thick, fibrous capsule with trabeculae extending in various ramifications throughout the entire organ. In the trabeculae and in the outer wall is a considerable amount of smooth muscle tissue which is under sympathetic nervous control. Therefore, it is an organ that is subject to easy contraction and relaxation, depending upon the various influences that affect the sympathetic nervous control mechanism. When the spleen is relaxed and there is no stimulation of its nervous mechanism, it is a large organ and contains a large amount of the blood of the vascular system. In the experimental animal this has been shown to be from one-fourth to one-sixth of the entire vascular content. The extent to which this blood is forced into the vascular system depends upon the amount of contraction of the splenic trabeculae.

Blood is brought to the spleen through a mesh of fine arterioles, and sluggishly flows through the splenic pulp, coming in intimate contact with the fixed splenic cells, after which it is collected into the venous sinuses, the walls of which are reticulo-endothelial cellu-

lar elements. Some of the arterioles open directly into the sinuses. The circulation is such that as the blood flows through the organ from the arterial to the venous side, it comes in intimate contact with the great mass of splenic pulp tissue. The blood flow is so slow that it is practically stagnant and appears as a vast lake, giving ample opportunity for the phagocytic mechanism to be extremely effective. Besides its reticulo-endothelial content, the spleen is a lymphoid organ presenting the usual malpighian corpuscles with their characteristic germinal centers. The function of the lymphoid elements is probably that of lymphoid tissue elsewhere, that is, the production of lymphocytes. To that extent, at least, it is a blood-forming organ.

The spleen has three major functions, namely, the destruction of red blood cells, the storage of large quantities of blood and the production of lymphocytes. It perhaps has other functions as well, such as the production of antibodies, and under certain abnormal conditions may itself become a hematopoietic organ producing new blood cells, as seen in certain types of leukemia where it is probable that islands of hematopoietic tissue are developed through the process of metastasis, as would be expected to be found in any malignant tumor.

It was chiefly the work of Barcroft,[3] who demonstrated that the spleen was an organ subject to relaxation and contraction, which could be caused by a variety of stimuli, and that there was great variation in the splenic volume of blood under variable conditions. Thus, the spleen can contract and force from it variable amounts of blood under conditions of excitement, fear, anger, increased muscular exercise, and any other condition characterized by an outpouring of adrenalin. As a matter of fact, the injections of adrenalin, pituitrin, acetylcholine and pilocarpine produce such a response in either the human or the experimental animal.

It has never been established whether or not the spleen produces any type of hormone. Some workers have suspected that the organ may produce a substance that is lytic for various types of blood cells, but this concept has never been established. Others have indicated their belief that perhaps the spleen produces some type of hormone, unknown as yet, that retards the cellular output of the bone marrow, but this concept still requires confirmation. Against the hormonal concept is the fact that the organ can be removed, and has been removed from large numbers of people, either because of disease or because of trauma; that in some people it is congenitally absent, and that all of this is not followed by any specific alteration that would indicate altered functions of other tissues or

organs in the body. The removal of a normal spleen, or its congenital absence, simply means that the individual lacks about one-fourth or one-fifth of his normal amount of lymphoid tissue, about one-fourth of his normal amount of phagocytic endothelial tissue, and that he has no splenic mechanism for the storage of large amounts of blood. It is well known, of course, that an individual can live very well without the spleen exerting these functions.

Studies of Ash-upmark[4] have shown that the splenectomized person should be considered normal, as his life expectancy is that of a normal person.

Hematologic Dysfunction of the Spleen

Of great interest in hematology is the fact that the spleen appears to be the greatest destructive organ for the red blood cells. The red cells are formed in the red marrow of the flat bones and, to some extent, in the epiphyseal ends of the long bones. The bone marrow, therefore, is a vast and far-flung organ located in some three hundred locations, constantly producing red cells in sufficient number to maintain the cellular equilibrium through the vascular system. Counterbalancing this is the destructive influence of the endothelial tissues of the spleen, the wear and tear of red cells with disintegration in the blood stream, and the possible destruction of cells that goes on elsewhere. There must be maintained, therefore, a normal balance between cell production in the marrow and cell destruction in the spleen. It is estimated that under normal conditions about 10,000,000 cells are destroyed each second, and of course, an equal number are produced by the bone marrow. It has also been estimated that the life of the normal red cell is from 25 to 50 days, perhaps about 30 days. At the end of this time the old, worn-out red cells, after being carried to the splenic vascular lake, are there presumably destroyed by a process of reticulo-endothelial phagocytosis. The endothelial elements of the spleen, therefore, appear to exert an uncanny selective action not only for the old worn-out red cells, but for other types of foreign material, including parasites, that may slowly percolate through the splenic pulp. If the phagocytic activity of the spleen is increased for any reason, or becomes altered in its capacity for this selective action and becomes unable to distinguish between normal red cells, white cells and platelets and those that are abnormal, then there results a condition that can best be designated as splenic dysfunction, characterized by excessive phagocytosis of various hematologic cellular elements.

In congenital hemolytic icterus the red cells are small, swollen, thick, and biconvex. The spleen destroys these in large numbers, regarding them, therefore, as abnormal cells in spite of the fact

that they still are capable of functioning well and carrying hemoglobin properly. It is also conceivable that the endothelium of the spleen becomes overactive for leukocytes for reasons that are unknown, resulting in the syndrome known as primary splenic neutropenia. This is probably the case in the disease essential thrombocytopenic purpura, where the blood platelets are looked upon by the spleen as cellular bodies to be removed from the circulation. Then it is further conceivable that phagocytic activity may become so increased that excessive destruction of any combinations of cellular elements may come about, even to the extent of excessive phagocytosis of red cells, platelets and neutrophils. These conditions have been designated by Doan and his associates[5] as primary splenic neutropenia if only the white cells are involved, and splenic panhematocytopenia if various combinations or all types of the cells are involved. Thus, the altered function of the phagocytic mechanism of the spleen may in one patient destroy the platelets in excessive numbers; in another patient, the red cells in excessive numbers; in another patient, the neutrophils in excess, and in still other patients various combinations of these three cellular types. All of this is based on altered or increased phagocytic function, resulting in a variety of clinical syndromes, the clinical picture depending upon the type of cell that is destroyed and the intensity of the process.

The reasons why the spleen develops this altered function are entirely unknown. In the process of destroying excessive numbers of blood cells, particularly if it is prolonged over a period of months or years, the spleen becomes enlarged and apparently becomes increasingly capable of exerting its perverted function in this respect because of its increased size and cellular volume. Therefore, it usually results in a progressive disease that at some time in the patient's life will most likely have to be interrupted, and the only sure method of interruption is removal of the spleen.

During the time that splenic function is altered the bone marrow is stimulated to further production of cellular elements to compensate for the loss due to increased cellular destruction. Therefore, the marrow becomes hyperplastic in its function to produce the type of cell that is being destroyed excessively by the spleen. Thus, in familial hemolytic icterus the marrow shows increased erythropoietic elements with vastly increased erythropoiesis, even to the extent of reconversion of normally inactive fatty marrow to actively producing red marrow. This is a form of compensatory functional activity of the marrow and as long as it is able to compensate for the increased splenic destruction, the patient most likely will not develop a syndrome characterized by anemia. This same process

exists in thrombocytopenic purpura in which there is compensatory growth of megakaryocytic tissue in the marrow in the attempt to produce increased numbers of platelets. Therefore, a fundamental principle in the diagnosis of these syndromes is the necessity for establishing the fact that the bone marrow of the patient is capable of producing cells at least to a normal extent. Bone marrow studies, therefore, are important in the evaluation of these splenic disordered states. In general, the criteria for the diagnosis of excessive splenic cellular destruction include the presence of an enlarged spleen, the lack of various cellular elements in the blood, and the presence of bone marrow that is capable of producing these cells to a normal extent.

If red cells are being destroyed in excessive numbers, the most reliable evidence of that fact, besides enlargement of the spleen, is the presence of products of red cell destruction in increased amounts. This can be determined by demonstrating increased amounts of bilirubin in the blood plasma, which is usually done by the finding of an elevated icterus index and by the demonstration of excessive amounts of urobilinogen in the urine. These two findings are fairly reliable indices that red cells are being destroyed in excessive numbers. The patient may actually be clinically or subclinically jaundiced. Coupled with this is the demonstration of increased bone marrow activity by finding excessive numbers of reticulocytes in the blood. Thus, on the one hand reticulocytosis is the most accurate indicator of accelerated erythropoietic activity in the marrow, and on the other hand increased bilirubin in blood plasma and urobilinogen in the urine are the most reliable indicators of accelerated destruction by the spleen. If the rate of destruction does not exceed the rate of production, the patient will not show clinical signs of anemia.

In instances of accelerated destruction of blood platelets, the clinical evidence is the presence of various types of hemorrhagic syndromes associated with thrombocytopenia. If other causes of thrombocytopenia can be ruled out with some degree of certainty, and if it can be demonstrated that the histologic pattern of the bone marrow is such that megakaryocytic tissue is present in normal amounts, then by a process of exclusion it can be assumed that the splenic function is altered to such an extent that platelets are being destroyed there in excessive numbers, such as one sees in the disease essential thrombocytopenic purpura.

If neutrophils are being destroyed in excessive numbers, then the clinical picture is one of granulocytopenia, and in such a patient, if it can be demonstrated that there is no other cause for the granu-

locytopenia, such as the ingestion of drugs, and if it can be further demonstrated that the bone marrow is capable of producing neutrophils in normal numbers, then also by a process of exclusion, one is justified in coming to the assumption that the splenic function is so altered that it is destroying these cells in excessive numbers. Such a syndrome is that described by Doan under the name of primary splenic neutropenia.

Diagnosis of Splenic Dysfunction

In every patient suspected of having one of the syndromes characterized by altered splenic function a careful history should be taken to determine whether or not the patient has been ingesting drugs or has been in contact with noxious and chemical agents that may be responsible for the blood cellular destruction and, of course, a physical examination to elicit findings that would uncover any underlying disease process. Certain laboratory findings should be made in every case, which are as follows:

1. Red cell count.
2. White cell count.
3. Estimation of hemoglobin.
4. Differential cell count.
5. Reticulocyte count.
6. Platelet count.
7. Measurement of bilirubin in the blood.
8. Measurement of urobilinogen in the urine.
9. Fragility test on red cells.
10. Studies of bone marrow on either aspirated or biopsied material.
11. In rare instances, studies on material removed by needle puncture of the spleen.

Needle puncture of the spleen is not always safe. Only recently I advised needle puncture of the spleen, after which the patient bled to death with massive peritoneal hemorrhage from the puncture hole, the death occurring some eight to ten hours later. This was not as tragic as might first appear since the patient had far advanced Hodgkin's disease of the spleen in its terminal stages, and the process would have terminated very shortly in any event. When needle puncture is done, it should be followed by careful and continuous observation of the patient for signs of shock and hemorrhage.

Surgeons are aware, of course, of the necessity for removal of the spleen in cases of hemorrhage following trauma, and also the

desirability of removal of the organ if a primary tumor is strongly suspected. In my series of over 80 splenectomies, there have been only two primary tumors, both of these being classified as hemangiomata.

Banti's Syndrome and Gaucher's Disease

The question of splenectomy in the so-called Banti's syndrome is one that cannot be decided in every case by following any flat rule, so it is a decision to be made after taking into account the various factors present in each patient. Some patients with this syndrome are not fit subjects for splenectomy, nor should the spleen necessarily be removed. This is especially true in elderly patients in whom there is evidence that the liver is involved to a rather marked degree. If there is evidence that the cirrhotic process in the liver is far advanced, and even though the spleen may be quite large, there is very little to be accomplished by removal of the organ in that type of patient, unless it is considered desirable to remove a massive spleen because of abdominal pressure and discomfort.

The best results from splenectomy in Banti's syndrome are those in which the spleen is removed from children and young adults and in which the splenomegaly is not associated with any considerable damage to the liver. In many patients these matters cannot be determined with any certainty unless it is done by exploratory operation, and at the operating table the decision can be made as to whether or not the organ should be removed. This decision is nearly always in the affirmative, based mainly on the consideration that the abdomen is already open and the opening is usually made with a view of performing splenectomy if necessary, and there is little to be gained by failing to remove the spleen after it is once exposed to view. From a practical standpoint, therefore, exploratory operation to determine whether the spleen should be removed is nearly always followed by removal of the organ. It is not known whether splenectomy in Banti's syndrome increases the life expectancy in the patient who has severe liver damage, but it is doubtful that it does.

Gaucher's disease is usually a clear indication for splenectomy provided it is not extremely far advanced, and the disease apparently does not exist elsewhere.

Congenital Hemolytic Icterus

This disease, sometimes called hemolytic jaundice, hemolytic anemia, chronic familial jaundice, or acholuric jaundice, is an inherited familial disease, characterized by prolonged or recurrent

attacks of jaundice with variable degrees of anemia and splenomegaly. It is a classic example of a true hemolytic anemia caused by destruction of excessive numbers of red cells in the spleen. The spleen exerts this action apparently because the red cells are inherently defective, the defect consisting of a partial swelling, and therefore spherocytic character, of the cell which makes it smaller, thicker and more rounded than the normal red cell. The agent that causes the partial swelling is unknown.

The defect is inherited as a true dominant according to the Mendelian law of heredity. It is seen frequently in successive generations in the same family. Hemolytic jaundice occurs in both sexes and appears to be more prevalent in white people, but I have recently studied the disease in a mulatto family, with one member being splenectomized with excellent results.

The type of red cell seen in the condition is known as a microspherocyte, and the condition microspherocytosis. There is no satisfactory explanation for it, but the fact that the cells apparently have already undergone partial hemolysis makes them more susceptible to further hemolysis by the action of hypotonic salt solution, so it is always characterized by an increased fragility test. Familial hemolytic icterus is the only disease in which the red cells show an increased fragility to hypotonic salt solution.

Even before birth the phagocytic mechanism of the spleen may become extremely active in the destruction of these defective cells, so that the finding of congenital hemolytic icterus in a newborn infant it not unusual. Usually the bone marrow of the infant has such reserve power that there is adequate compensation for the cellular destruction so that clinical evidence of the disease in the form of anemia becomes evident later, in early childhood or in adult life. In the meantime, however, the spleen gradually enlarges and this may be discovered at any time in a physical examination. Therefore, many cases of unsuspected familial hemolytic icterus are discovered by accident because of the unexpected finding of an enlarged spleen. Since the process of cell destruction has always been present, some patients show subclinical jaundice, and this is usually regarded by friends as the natural color of the patient.

The symptoms depend entirely upon the severity of the disease. Some patients go through their entire lives with ample bone marrow compensation, with only subclinical jaundice and develop no clinical syndrome that would lead one to suspect the disease. In the typical case, however, the jaundice is usually evident, the icterus index is elevated, and bilirubin in the blood plasma is increased in amount.

The patient may complain of weakness, fatigue and lassitude from anemia, or he may consult the physician because of the enlarged spleen. In rare instances the first evidence of the disease may be a so-called bone marrow crisis, in which the jaundice is deepened with an exacerbation of the process, and the anemia is profound with an associated leukocytosis. There may be fever, headache and variable pains, and the blood may show a leukemoid reaction simulating the blood picture of leukemia. The urinary findings are those of increased urobilinogen and if the degree of red cell destruction is severe, there may be actual hemoglobin in the urine. When patients are first seen in such a crisis splenectomy may have to be considered as an emergency measure, but most of them do not develop such crises. Complications may include chronic leg ulcers, and gallstones because of the excessive pigment in the biliary tract.

The essential diagnostic criteria consist of jaundice, enlarged spleen, microspherocytic anemia, increased fragility of red cells, and bone marrow that is hyperplastic. When such a picture is discovered, splenectomy is indicated and there is no other satisfactory treatment for the condition. The vicious circle of excessive red cell production and destruction can then be stopped only by removal of the spleen. Even after splenectomy, there is persistence of microspherocytosis. The fragility of red cells remains increased, but since the microspherocytes are capable of functioning as oxygen-carrying cells, and a large part of the phagocytic mechanism has been removed with the spleen, the load on the bone marrow is relieved, and in nearly every case the patient shows a clinical recovery which is permanent. Reticulocytosis may be quite marked before splenectomy, but subsides to normal after the spleen is removed. For some reason, unknown as yet, patients with familial hemolytic icterus may show the most unexpected and serious reactions to the transfusion of blood even though it is compatible by ordinary cross matching methods. Some deaths have been reported after transfusions from the use of blood that was known to be compatible. Therefore, transfusions should be given slowly with considerable caution, but every patient should have the necessary number of transfusions before splenectomy is performed.

I agree with the statement of Thompson[6] that "The symptoms of typical congenital hemolytic jaundice are promptly, completely and permanently relieved by splenectomy." This has been true in my own series of 32 patients, a group in which I could be absolutely certain of a correct diagnosis. After splenectomy there is an immediate rise of the red cells, and the platelets are unaffected, except in occasional instances when there may be a transient throm-

bocytosis. The leukocytes usually increase in number and may remain elevated for some weeks to several months after splenectomy. Also, there is usually a mild generalized lymphadenopathy which may be a compensatory increase of lymphoid and reticuloendothelial elements because of the loss of this tissue in the removal of the spleen.

Essential Thrombocytopenic Purpura

This disease, sometimes called purpura hemorrhagica or Werlhof's disease, is one of unknown cause, characterized by variable degrees of hemorrhages from skin and mucous membranes; a marked decrease in the number of platelets in the circulating blood, presumably because of increased destruction by the spleen; a prolonged bleeding time; a normal coagulation rate; a failure of the blood clot to retract; and a course of remissions and relapses, with eventual cure in most patients after splenectomy.

The disease occurs in both negroes and whites, with no essential difference in the clinical picture. It is mainly a disease of young people, the average age of reported cases being about twenty years, and it is rare in elderly people. It appears to be more common in females than in males, with a ratio of about four to one. The etiology is unknown, in that the cause of the platelet deficiency has not been clearly established. It is presumed to be on the basis of excessive splenic action, but the more fundamental reason, as to why the spleen exerts this action on the platelets, is entirely unknown. The one consistent finding is the decreased number of platelets which are sufficiently decreased to cause the resulting hemorrhages. The extent of the platelet reduction is by no means uniform since some patients present hemorrhagic syndromes with platelet counts that are much higher than others in whom there are no hemorrhages. A study of the spleens that have been removed from 24 cases of thrombocytopenic purpura in my own series fails to reveal any histologic changes that would throw any light on the disease. It is not necessary that the spleen be enlarged, and it was not enlarged in approximately one-half of my cases. The question as to whether or not the spleen produces an extract which destroys blood platelets remains unsettled.

The symptoms and clinical findings vary, being dependent upon the extent of reduction in number of platelets and the capacity of the patient to bleed. The history is usually that of previous and repeated episodes of bleeding, which have developed without apparent cause, interspersed with periods of remission. The type of hemorrhage may be nosebleed, postoperative bleeding, prolonged

menstruation, hematuria, cerebral hemorrhage, etc., but nearly always purpura. Examination usually reveals evidence of old purpuric spots or some that are present at the time of examination. Blood findings are characterized entirely by the marked diminution in the number of platelets. The red cells are usually normal but may be below normal if bleeding has recently occurred. The white cell picture is usually normal, but the number of cells may be slightly elevated if bleeding has recently occurred. The number of platelets may be as high as 100,000 or down to practically the vanishing point. The coagulation time is normal.

Before establishing a diagnosis of thrombocytopenic purpura, it is very important that all other causes of thrombocytopenia be eliminated if possible. Thus, it is important to determine whether or not the patient may have ingested or been in contact with drugs, chemicals or poisons that would be responsible for the platelet deficiency. For example, the analgesic agent sedormid is known to be capable of producing thrombocytopenia and other similar analgesic agents may be capable of this same action. It is now known that the sulfonamide drugs can also produce marked depression of the platelets in an occasional patient, presumably on the basis of impairment of bone marrow function. I have reported several such cases, and in my own series it seemed to be an irreversible process with death resulting from intractible hemorrhages in all patients.

The second important consideration is to be certain that the platelets are not low because of an incapacity of the bone marrow to produce them. This should be determined by bone marrow studies, with emphasis placed on the number of megakaryocytes and their morphologic normality, as these are the best criteria available for estimating bone marrow function with respect to its ability to produce platelets. If the patient has such a normal bone marrow and if he has not been subjected to the influence of drugs known to produce platelet deficiency and if the clinical picture is sufficiently severe, then splenectomy is indicated.

Some patients have this disease with a mild course extending over a period of years with minor episodes of bleeding which are not particularly important or distressing. Such patients should be observed over long periods of time before the final decision is made with respect to splenectomy. If they show spontaneous improvement through the years, then, of course, the operation should not be done, but if the disease appears to grow worse, then with each episode of bleeding splenectomy has to be considered more seriously.

After removal of the spleen there is usually an immediate im-

provement in the patient and bleeding appears to stop almost instantaneously. Dr. Lon Grove, of Atlanta, Georgia, who has performed most of the splenectomies in my series, feels that the bleeding that occurs during closure of the patient is far less than during exposure of the spleen. There is an immediate postoperative rise in the number of platelets and convalescence is usually quite uneventful. I have had three patients who apparently presented all of the classic characteristics of essential thrombocytopenic purpura who did not obtain complete relief from the operation for reasons that I am unable to explain. Patients in whom the clinical course is not so severe can be treated with medical measures such as transfusions when indicated, careful search for foci of infection, and small doses of radiation to the spleen. I have seen several such patients treated with such measures but most of them must eventually have splenectomy for permanent relief.

Primary Splenic Neutropenia

This syndrome, which was first described by Wiseman and Doan in 1942,[5] is characterized by a marked decrease in number of circulating neutrophils, with an enlarged spleen, and a bone marrow that gives evidence of being normal in its capacity to produce neutrophilic leukocytes. As pointed out earlier in this paper, the existence of the syndrome is based upon the presumption that the spleen is capable of exerting a selective affinity in its increased phagocytic function for the neutrophilic leukocytes, presumably destroying these cells as they pass into the splenic reservoir so that they are constantly depleted in the peripheral blood.

When the physician is confronted with any patient with neutropenia, certain diagnostic possibilities must be borne in mind. First, the importance of a careful history cannot be overemphasized to be certain that the patient has not been receiving drugs capable of damaging the bone marrow to such an extent that it fails to produce the normal number of neutrophils. Such drugs are now known to include amidopyrine and its various combinations, the organic arsenical compounds, the sulfonamide drugs, and perhaps others. If it can be determined with some assurance that the neutropenia is not on the basis of drug administration or of exposure to chemicals, then one should consider diseases that may be accompanied by neutropenia, such as aplastic anemia and aleukemic forms of leukemia. These, of course, are excluded on the basis of other hematologic findings. Finally, by a process of exclusion, if the patient has an enlarged spleen, primary splenic neutropenia can then be suspected. Before considering splenectomy, it is necessary

that it be demonstrated clearly, if possible, that the bone marrow is normal with respect to its capacity to produce neutrophils. This can be done by examination of aspirated material from the marrow.

Only one who is skilled in examination of bone marrow is qualified to give an opinion on this point, but even the opinion of an expert is based to some extent on presumption because of the limitation of the method. When these things have been done, however, and all other causes for neutropenia have been excluded, then a tentative diagnosis of primary splenic neutropenia is justified. After sufficient observation of the patient, splenectomy can then be done in an effort to relieve the neutropenic syndrome. I have seen only two patients in whom I have made this diagnosis. One was an elderly woman who presented a syndrome of chronic neutropenia of many months' duration, the leukocyte count varying from 3,000 to 3,500 cells per c. mm. She was moderately anemic and the platelets were not affected. Her spleen was enlarged, and apparently her bone marrow pattern was normal. After splenectomy this patient had a return of normal cellular values. The time required for the leukocytes to become normal was approximately four months, and she has remained in good health since. I have studied a second patient who apparently has this syndrome, who is a 26-year-old woman with what I have termed "walking neutropenia." We have applied this term to this particular patient since examination of her peripheral blood over a period of more than a year has never revealed the leukocytes to be over 2,000 cells per c. mm., and they usually range around 1,000 c. mm., with 90 to 95 per cent lymphocytes. During this time she has remained in excellent health, does her household duties in a satisfactory way, becomes only slightly fatigued on exertion, and has normal red cell values and normal platelet values. The astonishing thing about this patient is the fact that her neutrophils have remained at this low level for at least a year to our certain knowledge, and she still fails to reveal any clinical evidence of the hematologic defect. I consider her a case of primary splenic neutropenia, but as yet, because of her relatively good health, she has not consented to a splenectomy. It would be my prediction that when and if splenectomy is performed, it probably will be followed by an excellent therapeutic result, with restoration of neutrophils to their normal level.

Primary Splenic Panhematocytopenia

This syndrome, described by Doan[7] under the name of congenital splenic panhematopenia, is characterized by an excessive phagocytic function of the spleen with respect to any and all types of the cellu-

lar elements of the blood that are produced in the bone marrow. Thus, there may be combinations of thrombocytopenia and anemia, thrombocytopenia and neutropenia, neutropenia and anemia, or thrombocytopenia, neutropenia and anemia, with all of these being described with the single designation of "primary splenic panhematocytopenia." The term, therefore, is used to designate the group of syndromes resulting from splenic activity that affects two or more of the bone marrow cellular elements. In those patients showing depletion of all three of the marrow cellular elements, the most important differential diagnosis is to differentiate this from aplastic anemia or hypoplastic anemia in which the bone marrow is unable to produce sufficient numbers of these cells. The most important studies to be made in establishing this diagnosis are those designed to demonstrate the functional capacity of the marrow. If it can be shown that the marrow is capable of producing these cells in adequate numbers and if the spleen is enlarged, then one is justified in making a presumptive diagnosis of splenic panhematocytopenia. I have seen three such patients who apparently had variations of this condition. One was a middle-aged woman who, for many months, had presented the syndrome of severe hemolytic anemia and thrombocytopenia. The anemia was hemolytic as shown by the fact that the reticulocyte count was consistently high, the products of red cell destruction were consistently increased in the blood and urine, and the bone marrow was hyperplastic with respect to its erythropoietic pattern. Furthermore, there could be demonstrated no history of exposure to drugs, chemicals or toxins that would produce this syndrome. Consequently, on the basis of these findings splenectomy was recommended and performed with excellent results and fairly prompt restoration of red cell and platelet values. I have studied another patient who showed a marked depletion in the number of platelets with an associated hemorrhagic syndrome, a marked leukopenia in which the total number of granulocytes in the blood usually ranged around 2,000 cells per c. mm., with an enlarged spleen and apparently a normal bone marrow pattern. This patient also was splenectomized with excellent results, and she remains well at this time, three years after splenectomy. I have recently studied a third patient, a middle-aged woman, who, for several weeks after an acute onset, presented a consistent thrombocytopenia and marked hemolytic anemia with no disturbance of the leukocytes. The anemia was hemolytic, the reticulocytes were 25 per cent, there was some suggestion of microspherocytosis, and also a questionably increased fragility test. There was no evidence of excessive cellular destruction as measured by bilirubin content of plasma or by increased urobilinogen in the urine. The spleen in this patient was only ques-

tionably enlarged. I recommended splenectomy, and after a stormy postoperative course her cell values have been restored to normal.

The above three instances represent syndromes that I would classify under the designation of splenic panhematocytopenia in which two or more cellular elements were apparently being destroyed in excessive numbers by the spleen. These are syndromes that in the past have been probably falsely attributed to inability of the bone marrow to produce these cells, and have been designated by some workers, I am sure, as hypoplastic anemia.

Even though there may be evidence that there is a partial inhibition of bone marrow output of these cells, this within itself does not rule out splenectomy as a possible therapeutic aid. It is quite possible, of course, that the bone marrow cellular output may be sufficient to maintain normal levels in the blood, provided the normal destructive influence of the spleen is removed. In such patients splenectomy restores the rather delicately balanced cellular equilibrium in the blood, and if the primary objective of restoring normal blood values is attained, then the operation is decidedly worthwhile.

Conclusions

1. It is becoming clear that the indications for splenectomy should be considerably widened to include a number of syndromes that are characterized by excessive phagocytic activity of the spleen.
2. The syndromes that appear to be caused by excessive splenic activity include congenital hemolytic icterus, essential thrombocytopenic purpura, primary splenic neutropenia and primary splenic panhematocytopenia.
3. In every case of suspected splenic phagocytic overactivity the red cell depletion should be proved to be on a basis of accelerated red cell destruction with attempts at bone marrow compensation, and in the neutropenic syndromes possible causation by ingestion of drugs must be definitely ruled out.
4. In nearly all of these syndromes the spleen is enlarged to a variable degree, with the possible exception of essential thrombocytopenic purpura.
5. In all of these syndromes it should be clearly demonstrated, if possible, that the bone marrow is capable of producing these cells in normal numbers.
6. On the whole, the results of splenectomy in this group of diseases have proved to be quite successful.
7. It is highly desirable that our surgeons recognize the increased

usefulness of splenectomy as a therapeutic agent in this group of diseases characterized by depleted cell values in the blood and caused by overactivity of the spleen.

REFERENCES

1. Kracke, R. R.: The Effect of Splenectomy in the Purpuric Diseases, South. Surgeon *2:* 203, 1933.
2. Dameshek, W.: Editorial, Blood *1:* 173, 1946.
3. Barcroft, J.: Alterations in the Volume of the Normal Spleen and Their Significance, Am. J. M. Sc. *179:* 1, 1930.
4. Ash-upmark, E.: Remote Effects of Removal of the Normal Spleen in Man, Svenska läk.-sallsk. handl. *61:* 197, 1935.
5. Wiseman, B. K., and Doan, C. A.: Primary Splenic Neutropenia: A Newly Recognized Syndrome Closely Related to Congenital Hemolytic Icterus and Essential Thrombocytopenic Purpura, Ann. Int. Med. *16:* 1097, 1942.
6. Thompson, W. P.: Hemolytic Jaundice: Its Diagnosis, Behavior and Treatment: A Review of Forty-five cases, J. A. M. A. *107:* 1776, 1936.
7. Doan, C. A., and Wright, C. S.: Primary Congenital and Secondary Acquired Splenic Panhematopenia, Blood *1:* 10, 1946.

PARATHYROID CYST

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CYSTIC tumors of the neck constitute a group of lesions which are interesting in their embryologic implications and may present difficult diagnostic problems. Among these are branchiogenic cysts, thyroglossal cysts, hygromas, various cysts of the thyroid gland, sebaceous cysts and other cystic lesions of less frequent occurrence. True cysts of the parathyroid glands, certainly those of sufficient size to be of clinical significance, are almost unheard of.

Case Report

A 17-year-old high school boy complained of hoarseness and moderate dyspnea on exertion of approximately 2 months' duration. He had known for "some time" that there was a "lump in the left neck." He had no other complaints. Examination revealed a well-developed young man who appeared healthy. There was a hard, round tumor in the region of the lower pole of the left thyroid lobe. It was about the size of a golf ball, slightly movable, and had displaced the trachea moderately to the right. Examination was otherwise entirely negative. Blood pressure, pulse, pulse pressure and heart action were all within normal limits. Basal metabolism was not determined since it was obvious the lesion was non-toxic. Blood chemistry studies were not made as there were no clinical indications for them. A clinical diagnosis of non-toxic adenoma, probably fatal adenoma, of the thyroid was made and surgical removal advised. At operation a peculiar type of pearly-colored cyst was found. It was intimately adjacent to, but apparently not a part of, the lateral aspect of the lower left thyroid lobe. The wall of the cyst was inadvertently ruptured just as it was enucleated and contained a pale milky material a little thicker than water. The character of this fluid was such as to give temporary apprehension that some type of dilation of the proximal end of the thoracic duct had been encountered. Such, however, fortunately, was not the case. The thyroid gland was carefully explored and found to be normal in size, color and consistency. Operative diagnosis was deferred pending a pathologic report. The patient made an uneventful recovery.

Pathologic Report

The wall of the cyst, which measures up to 4 mm. in thickness, is composed of parallel bundles of collagenous connective tissue characterized by a paucity of nuclei. Numerous vessels—capillaries and arterioles—are found within the wall. Also there are seen several small foci of lymphatic tissue scattered throughout the width of the cyst wall. No epithelial lining is found on the inner surface. Scant, flat endothelial-like cells are seen to form the inner lining of the cyst. Only in one area, throughout various blocks of the wall of the cyst, is there found an epithelial structure, which in its largest diameter

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measures 3 mm. It is situated in the innermost portion of the wall and protrudes as a small nodule into the lumen of the cyst. The bundles of connective tissue separate and fan out as they approach the nodule, but do not cover the latter at the inner surface of the cyst; the nodule of epithelial cells borders the lumen of the cyst directly.

The epithelial cells themselves are polygonal in shape and possess a vacuolic cytoplasm with sharply demarcated cell borders and centrally located, vesicular nuclei, which are relatively small in comparison to the cytoplasm. The latter does not possess a visible granulation. The epithelial cells are arranged

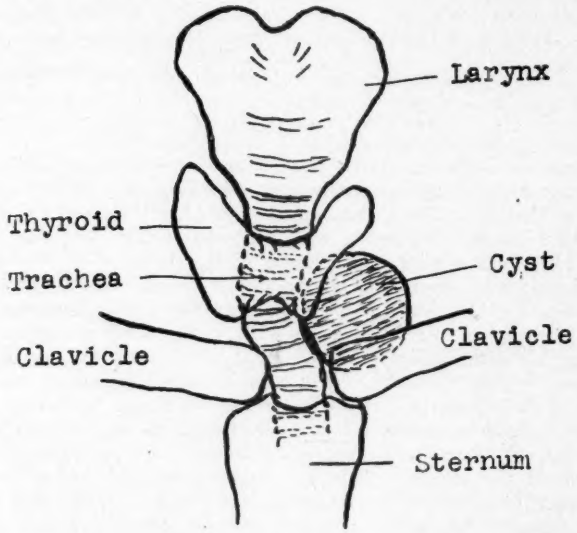

Fig. 1. Schematic drawing showing position of the cyst lying partly subclavicularly on the left and displacing the trachea moderately to the right.

in clusters and cords, separated from each other merely by a fine reticular network and numerous capillaries which are engorged. The nodule is rather well demarcated from the adjacent connective tissue structures.

No thyroid tissue or other epithelial structures are seen in the wall of the cyst.

The nodule bordering the lumen of the cyst is identical in its histologic structure with that of a parathyroid body, solely composed of so-called water-clear ("wasserhelle") cells. Since the nodule is not separated from the lumen of the cyst by any other structures, it is assumed that the cyst originated from a parathyroid body.[4]

Discussion

Tumors of the parathyroid glands are quite rare and usually consist of the so-called parathyroid adenomas or what has been termed "work hypertrophy." Cysts sufficiently large to give subjective or

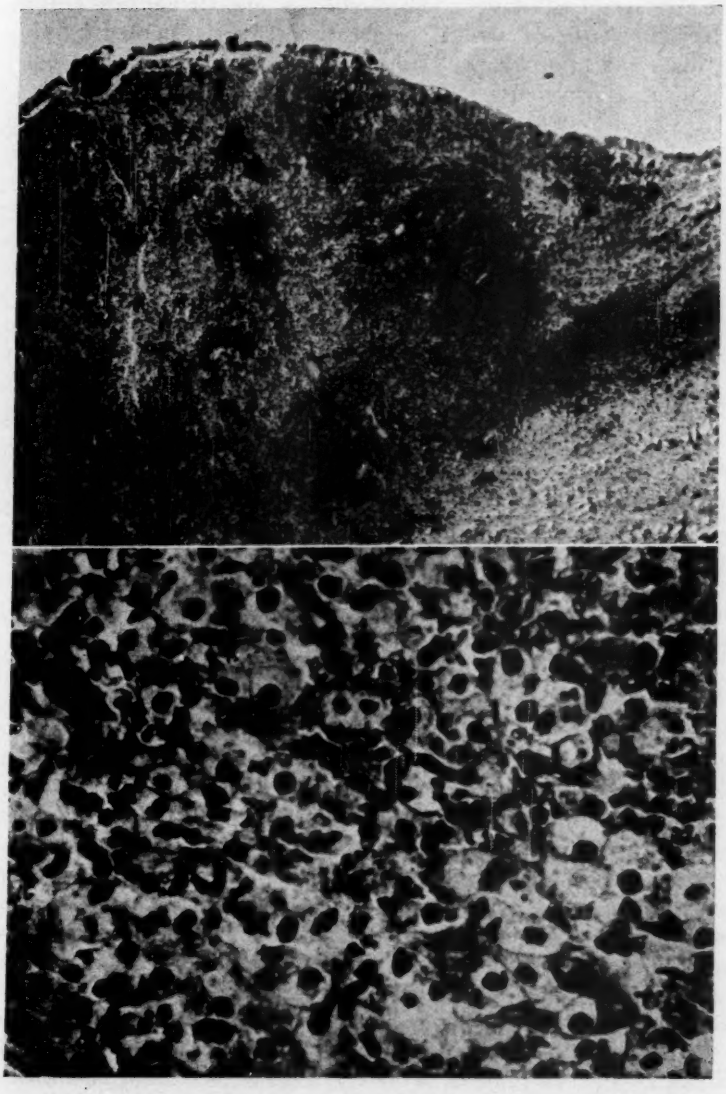

Fig. 2. Low power michrophotograph of the parathyroid gland and a portion (to the right) of the cyst wall. In the wall can be seen a small lymphatic tissue focus. The endothelial-like cells disappear over the parathyroid. There is no evidence of small cyst formation within the gland.

Fig. 3. High power michrophotograph showing typical parathyroid cells—the so-called water-clear (wasserhelle) cells. There is no evidence of follicular cyst formation.

objective symptoms have not been reported in the English literature, so far as has been possible to ascertain. Goris, quoted by Guy, DaCosta and others, in 1905 made a diagnosis of cystic degeneration of a parathyroid gland on a tumor removed from the neck of a 23-year-old male. It consisted of three cysts, closely connected, but independent of and not attached to the thyroid gland. Microscopically, it revealed encapsulated colloid and degenerated parathyroid tissue.[2] Undoubtedly small cysts discovered on microscopic examination are of not infrequent occurrence. To quote from Herxheimer: "Cysts are very common in parathyroid bodies. Petersen found them six times in one hundred cases; von Verebely in twelve per cent of his cases, and similar reports are made by Moller, Erdheim and recently Danisch. I have seen them myself repeatedly. Already Kohn has spoken of their occurrence. It is difficult to state whether the cysts are congenital malformations or due to changes in later life. However, the fact that they increase in number in older individuals points to the probability that the cysts grow in the course of life and only then do they produce an atrophy of the parenchyma of the gland by pressure. The conditions probably are similar to those in the kidney in that minute developmental abnormalities later on grow into larger cysts. The cysts are filled with a light, hardly stainable content in which desquamated fatty epithelial cells or colloid material is found, or they may be filled with a colloid content. In any case they grow by retention of the secretion. They may originate from follicles. Such cysts may grow so large that they may replace almost the entire gland or several of them together may replace the entire organ. These are probably cases of cystic degeneration. As in the kidney one can draw a line from small insignificant single cysts to such formations which deserve the term of cystic parathyroid bodies. An almost complete replacement of glands by cysts has been reported by Schaper in the sheep, Litty in the horse, Rossi in the cow, Aglagna in the dog, Petersen found them in human beings. Harvier speaks of transformation vesiculaire. Erdheim of cystomata of the same formation. Thompson and Harrison distinguish between retention cysts, polycystic degeneration and branchiogenic cysts. There are cysts which are lined by ciliated epithelial cells which have been seen by Edmunds, Kohn, Nicolas and others in the cat, Lusena in the dog, Danisch in his cases in human beings, and it is this type of case which points to developmental abnormality. To this latter type of cyst certainly belong the cysts which are found in juxtaposition to the parathyroid body, although the latter may be partially or completely embedded in its wall, cysts which have been classified as branchiogenic cysts. . . ."[3] Nylander[5] in 1929, reported a "Parathyroid Cyst of the

Neck," but neither the periodical nor a translation has been obtained.

It would seem, therefore, that this case is probably the third reported in the literature, that is, of a parathyroid cyst of sufficient clinical significance to give objective and/or subjective symptoms. It is difficult to classify this cyst. It was filled with a pale milky material which likely contained desquamated cells and maybe some colloid material. The wall was composed of connective tissue and lined with endothelial-like cells, which, of course, were not ciliated. Is it a retention cyst, polycystic degeneration or a "pinched-off" branchiogenic cyst? The fact that some lymphatic tissue is present might suggest its branchiogenic origin. Branchiogenic cysts are lined with epithelium, which under pressure, might become flattened into endothelial-like structures. It is *not* the opinion of the author that this is of branchiogenic origin, but it might be somewhat difficult to disprove it! Apparently the small millimeter-or-so sized cysts reported to be of frequent occurrence are only of academic interest, for it is not recorded that there are any larger than a normal sized parathyroid gland. There is no evidence that they affect parathyroid metabolism.

There was no evidence in this case that parathyroid metabolism was in any way affected. The symptoms were only those of a tumor in the neck and subsequent tracheal pressure manifestations.

Development of a Parathyroid Cyst

It is hard to give an adequate explanation of why cysts should develop in the parathyroids, or why (certainly large ones) are of such extreme rarity. As stated, it is not infrequent for presumably normal parathyroids to have some follicles containing colloid somewhat similar in appearance to that in the thyroid glands. It is conceivable that one of these follicles might become greatly distended resulting in the cystic parathyroid. The embryologic development of the parathyroids is worthy of serious thought as a possible explanation of the cause of this cyst. The superior parathyroids develop as solid outgrowths from the dorsolateral walls of the fourth pharyngeal pouches, and the inferior parathyroids develop as solid outgrowths from the rostral walls of the third pharyngeal pouches. It may be that infrequently one or the other of these solid outgrowths might carry along an extension of the lumen of the pharyngeal pouch. This pouch then might continue to accumulate fluid and so cause a cyst to develop.[1]

Conclusions

Parathyroid cysts sufficiently large to give objective signs and symptoms are exceedingly rare. Such a case report is presented, together with a discussion of the pathology and possible sources of development. Parathyroid metabolism is not affected.

REFERENCES

1. George, W. C. Personal communication.
2. Goris. J. de chir. et ann. Soc. de chir. 584, 1905.
3. Herxheimer, Gotthold. Translation from Henke-Lubarsch, *8:* 598.
4. Kimmelsteil, Paul. Personal communication.
5. Nylander, P. E. A. Parathyroid Cyst of the Neck, Acta chir. Scandinav. *64:* 539-542. 1929.

THE MANAGEMENT OF COMMON DUCT STONE MISSED AT OPERATION

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THE treatment of a common duct stone overlooked at the time of operation is usually a difficult problem. This is so largely because the factors which make overlooking the stone more likely also operate to make its subsequent management more difficult. Some of these factors are: Obesity; abnormal relationships of the structures in the hepatoduodenal ligament; obstructive jaundice with its frequently associated poor general condition; dilatation or thickening of the hepatic and common ducts; and poor anesthesia. It is evident that these factors may operate to make the management of the overlooked stone more difficult, particularly if it must be approached surgically.

It is probable that a surgeon of wide experience will rarely overlook a stone in exploring the common duct. That this can happen, however, is attested by the reports in the literature.[1,2,3,4,5,6,7,8,9] There are, no doubt, many unreported cases, and many other cases in which the stone is overlooked, only to be removed several years later, or to remain and cause episodes of "post-cholecystectomy colic."

The realization that a stone has been missed usually occurs when, upon clamping the "T" tube, the patient experiences pain and leakage of bile about the tube. These symptoms make their appearance if the stone is in the distal portion of the duct in the region of the papilla, and it is causing a high grade of obstruction. This is not necessarily the case if the stone is in the proximal portion of the duct, or in one of the hepatic ducts. It is possible that it may be discovered in this location if a postoperative cholangiogram is performed. Such an incident occurred in one of the cases herein reported.

The postoperative cholangiogram depicted (fig. 1) is that of a jaundiced white male who had been subjected to exploration of the common duct and cholecystectomy. Several stones had been removed from the common bile duct in the region of the ampulla of Vater, and a "T" tube inserted for drainage. The tube had been clamped postoperatively without pain or leakage and, since the

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lipiodol had been seen to pass on into the duodenum, the tube was in due course removed. His biliary fistula promptly healed. He returned one year later with a history of biliary colic, associated with chills, fever and jaundice. At operation a stone was removed

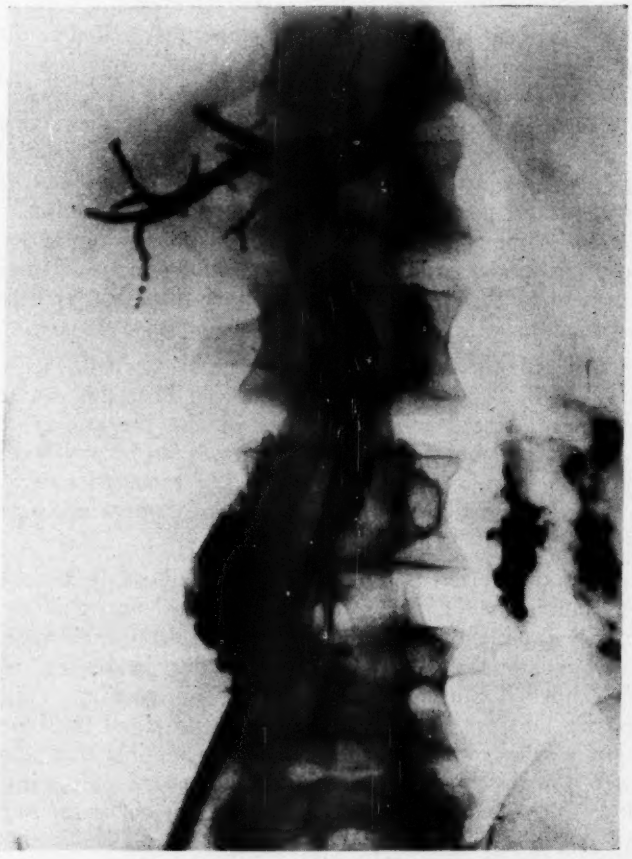

Fig. 1. Postoperative cholangiogram demonstrating defect in common hepatic duct caused by overlooked stone. Patient returned several months after removal of "T" tube, with a history of biliary colic and jaundice. At operation a stone having the same configuration as the defect noted in the cholangiogram above was removed from the pancreatic portion of the common bile duct.

from the pancreatic portion of the common duct, and its physical appearance tallied with the defect noted in the cholangiogram shown in Figure 1. This defect is best seen in the picture as that one lying in the interspace between the second and third (reading

from the top) vertebral bodies. The significance of this defect had not been properly evaluated or understood at the time it was originally noted. The patient has subsequently remained well.

The other figures represent a series of postoperative cholangio-

Fig. 2. Postoperative cholangiogram in second patient following first operation. Note failure of the duct system to fill and the rapid expulsion of the lipiodol into the duodenum. Such a finding may indicate stones in the proximal portion of the biliary tree. After removal of the tube a biliary fistula persisted.

grams made during the course of the treatment of a 54 year old white woman. This patient was admitted to the hospital deeply jaundiced, the jaundice being of several weeks' duration. At operation the gallbladder was found to be an atrophic remnant; the remainder of the biliary tree was markedly dilated and its walls

moderately thickened. The common bile duct was explored through an incision in its supraduodenal portion, but no stones could be found. The sphincter of Oddi was dilated with a 4 mm. probe and a "T" tube was inserted in the duct for drainage. Figure 2 repre-

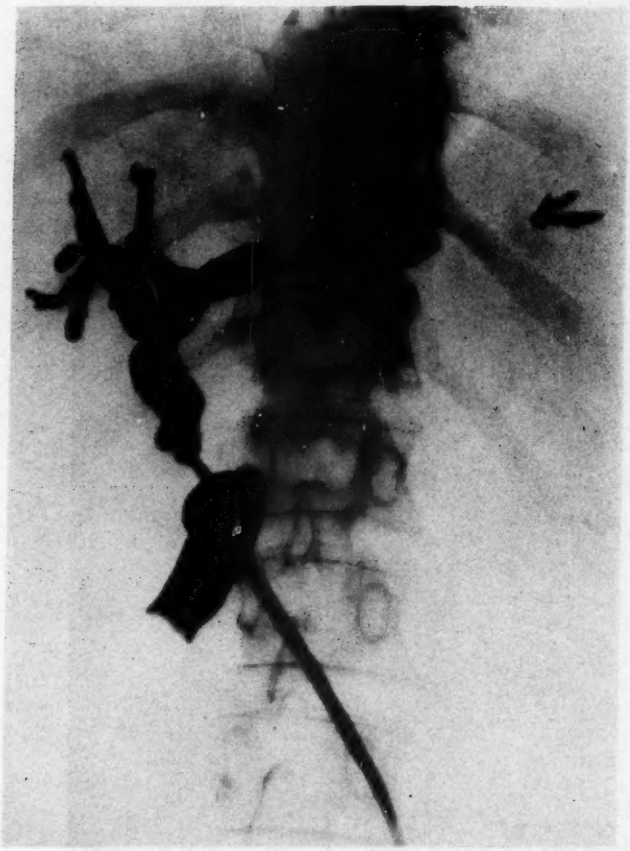

Fig. 3. Cholangiogram following second operation in this patient. One large round stone had been removed. Note complete obstruction of the duct, and the character of the shadow cast by the remaining stone in the pancreatic portion of the duct.

sents a cholangiogram following this, the first operation. Note that the biliary tree cannot be filled and that the lipiodol is rapidly expelled into the duodenum. Clamping of the tube at this stage did not cause any distress or leakage of bile, and the stools would become brown in color. The tube was removed but the biliary fistula would not close. The common duct was again explored surgically,

and a large round stone measuring about .75 cm. in diameter was removed from the pancreatic portion of the common bile duct. Figure 3 depicts the cholangiogram following this, the second operation. Note that complete obstruction exists in the distal end of the

Fig. 4. Appearance after instillation of ether through "T" tube into the common duct. Close inspection reveals shadows suggestive of a fragmented stone, the fragments being coated with lipiodol.

common bile duct, and that the entire duct system is dilated. Also, note the inverted meniscus-shaped deformity of the common duct, somewhat resembling the calyx of the kidney pelvis.

The patient had never been in very good condition, and at this time was considerably debilitated. It was decided to attempt frag-

mentation of the stone by instillations of ether into the "T" tube as described by Pribram, Walters, and others. Figure 4 illustrates the first cholangiogram made after several instillations of ether, and mixtures of ethyl alcohol and ether. Complete obstruction is

Fig. 5. Cholangiogram made after additional instillation of ether into the common duct Note that lipiodol passes into the duodenum but that the duct system remains dilated

still seen to exist but close inspection reveals several shadows just inferior to the distal end of the duct which suggest fragments of stone coated with lipiodol. Additional instillations of ether in the "T" tube were made. Figure 5 shows the cholangiogram after these. The radiopaque substance is seen passing into the duodenum; some dilatation of the duct system persists. Figure 6 shows the

condition of the duct system some three weeks later. The "T" tube was removed at this time, the wound promptly healed, and the patient has since remained well, a period of over 2 years.

These cases illustrate that postoperative cholangiograms can

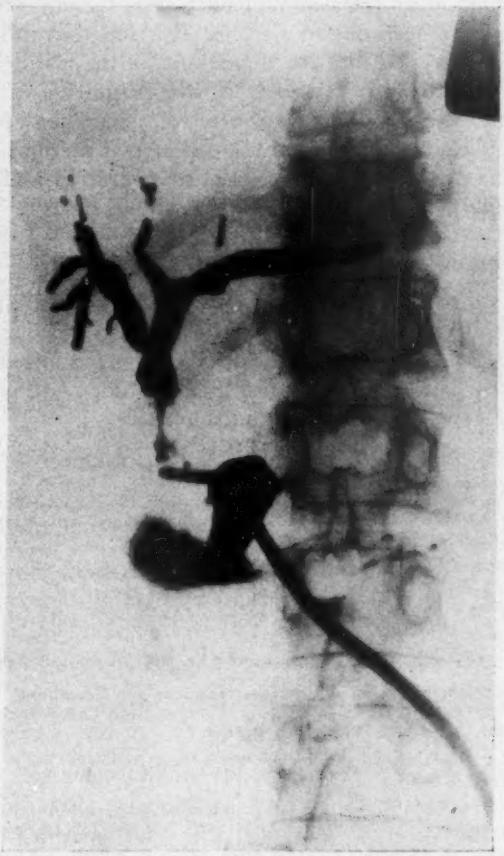

Fig. 6. Final appearance just before removal of "T" tube. The biliary fistula promptly closed, and patient has remained well for a period of over two years.

furnish valuable and interesting information concerning the condition of the biliary duct system. The first case, as illustrated in Figure 1, is interesting in that our lack of experience with postoperative cholangiography caused us to overlook a shadow which subsequently was proven to have been caused by a stone. It is interesting to note that in the second case (fig. 2) the biliary ducts

could not be filled, and the lipiodol was rapidly expelled into the duodenum. One wonders whether or not this irritability of the ducts was due to the remaining stones, and if one should not suspect such a pathologic lesion when such phenomena are exhibited during the course of cholangiography.

The obstruction of the common duct (figs. 3 and 4) seems undoubtedly to have been due to a stone. It is felt that this stone was either forcibly expelled into the duodenum by the instilled ether, or that it was fragmented or dissolved, the fragments or the solute passing on into the intestinal tract, and releasing the obstruction of the common bile duct. It would seem that this method deserves a trial in the treatment of stones missed at operation and remaining in this portion of the duct.

SUMMARY

Two cases of common duct stone missed at the time of operation and subsequently demonstrated by postoperative cholangiography are reported. In one case the overlooked stone was apparently fragmented by instillations of ether into the "T" tube and the fragments subsequently expelled into the duodenum.

REFERENCES

1. Walters, W., and Wesson, H. R.: Fragmentation and Expulsion of a Common Duct Stone Into the Duodenum by Using Ether and Amyl Nitrite, Surg., Gynec. & Obst. *65:* 695-697 (Nov.) 1937.
2. Pribram, B. O.: New Methods in Gallstone Surgery, Surg., Gynec. & Obst. *60:* 55-65 (June) 1935.
3. Morton, H. B.: The Treatment of Common Duct Stone Missed at Operation, Surgery *12:* 591-598 (Oct.) 1942.
4. Burgess, C. M.: Solution of Gallstones, J.A.M.A. *114:* 2372-2373 (June 15) 1940.
5. Best, R. R.: Cholangiographic Demonstration of the Remaining Common Duct Stone and its Non-operative Management, Surg., Gynec. & Obst. *66:* 1040-1046 (June) 1938.
6. Raffl, A. B.: Experimental Studies on the Solvent Action of Ether on Gallstones, Am. J. Surg. *52:* 65-69 (April) 1941.
7. Probestein, J. G., and Eckert, C. T.: Injection of Ether Into the Biliary Tract as Treatment for Choledocholithiasis, Arch. Surg. *35:* 258-267 (Aug.) 1937.
8. Barnes, J. P.: Probing the Common Duct Through a T-tube Four Weeks Postoperatively, South. Surgeon *11:* 35-41 (Jan.) 1942.
9. Pribram, B. O.: Ether Treatment of Gallstones Impacted in the Common Duct, Lancet *1:* 1311-1313 (June 10) 1939.

ECLAMPTOGENIC TOXEMIA

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A FINAL classification of the late toxemias of pregnancy has not been made. Stander suggests a classification including "low reserve kidney," chronic nephritis of pregnancy, preeclampsia, and eclampsia. There are many patients who have symptoms of toxemia of mild degree which, according to Stander, fall into the group of "low reserve kidney" type which is characterized by decreased urinary output, increase in body weight, and the presence of a small amount of albumin in the urine, with or without an elevation in blood pressure. As the symptoms increase, they are those of preeclampsia such as elevation of blood pressure, more marked edema, headache, dizziness, increase of weight, nervousness, and excitability. It would seem that "low reserve kidney," preeclampsia, and eclampsia are stages of the same process. We are following the classification recommended by the American Committee on Maternal Health.

PREECLAMPSIA AND ECLAMPSIA

Inasmuch as the latter is an exaggerated form of the former, these two conditions are considered together.

Definition: A disease of pregnancy probably due to ovular toxins, perhaps associated with nutritional deficiencies, and characterized by distinct pathologic lesions and a syndrome of symptoms, the most important of which are convulsions.

Varieties: We speak of preeclampsia in the early stages, before convulsions occur, and of eclampsia in the presence of convulsions.

Etiology: Notwithstanding the many studies as to the cause of this disease, it remains unknown today. The most logical theory is that these two conditions are one form of toxemia, causing, or caused by, perversions in metabolism and that a toxin circulating in the blood stream upsets the water balance, causes liver changes, directly or indirectly, and degenerative changes in the kidneys, and that convulsions occur, due to direct toxic action of the cerebral cortex, pressure caused by edema, or vasospasm affecting the brain.

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It would be erroneous for one to say that the cause of eclampsia is known. On the other hand, observation and research have added sufficient information to change considerably our conception of the cause of this condition. It was first noted by the ophthalmologists that small blood vessels in the retina were constricted in the presence of preeclampsia. It had long been known that the urine volume was decreased, and the degree corresponded to the severity of the disease. There were those who recognized that the decrease in urine volume was also a matter of vasoconstriction. It became apparent that this constriction of the capillary bed was generalized, not only in the eye and kidney, but throughout the body and accounted for the rise in blood pressure. It had also been noted that the duration of the hypertension of preeclampsia was more important than the severity of the disease—that is, that the patient who had a moderately elevated blood pressure for several weeks would suffer permanent kidney damage, even though there had been no convulsions; whereas in a rapidly developing toxemia of severe degree lasting only a few days and attended with convulsions, there might be no permanent kidney damage. These clinical observations were supported by experimental evidence. Many believe that the placenta produced a pressor substance in preeclampsia, but it could be reasoned just as well that a pressor substance is always present during pregnancy and that a vasorelaxor ordinarily present in normal pregnancies is absent in preeclampsia. It may be that constriction of the small blood vessels alters the vessel wall sufficiently to aid in the production of edema.

Another factor must be considered, at least in certain regions. We have found that the nutritional factor is highly important, and there must be sufficient concentration of the serum proteins in the blood stream to prevent fluid from becoming extravascular.

Pathology: Liver—Necrosis in the periphery of the lobule extending toward the center usually immediately surrounding the portal vein. Hemorrhages occur in the lobules and are also frequently found beneath the capsule of the liver. A yellowish appearance is due to fatty degeneration.

Heart—Fatty degeneration of the myocardium.

Brain—Edema and hemorrhagic foci.

Skin—Edema, jaundice, occasionally subcutaneous hemorrhages.

The pathologic changes which have been mentioned are probably all secondary, and the primary change is in the placenta. There are those who are able to detect the eclamptic placenta by histologic changes.

Symptoms: Edema, although slight, is often seen in the latter part of pregnancy due to interference in circulation. The presence of any edema should suggest toxemia of pregnancy.

Persistent headache is a constant symptom.

Spots before the eyes.

Twitching of the face and pain in the epigastrium are commonly seen before the onset of convulsions.

Hemorrhagic retinitis is not constant but frequent.

Urine: Albumin is found in varying amounts. The amount of albumin is not always indicative of the severity of the toxemia. The quantity of urine is usually decreased. The appearance of casts usually indicates that the disease is progressing. Blood may be found in the urine in severe cases.

Blood pressure: A rise in blood pressure is a significant sign. Blood pressure readings above 135 mm. suggest the presence of toxemia. The blood pressure may rise, however, to as much as 250 mm. or higher. Convulsions may occur either antepartum, intrapartum, or postpartum. It is hardly necessary here to describe the typical convulsion.

Diagnosis: The diagnosis is established from the signs and symptoms as listed above and, in the presence of convulsions, must be differentiated principally from epilepsy, hysteria, uremia, and all forms of coma.

Treatment of non-convulsive toxemia (preeclampsia): Prenatal care has done much to eliminate the incidence of these conditions. There is no better way to treat the disease than to prevent it by frequent prenatal visits. One should adopt the attitude that every pregnancy is a potential toxemia. Routine examination of the urine should be done in every case at least every two weeks during the second half of pregnancy. The routine examination of blood pressure should be done at regular intervals. A blood pressure of 135 mm. should be considered as an indication for active treatment and continued rise in blood pressure should lead the physician to watch the case very carefully.

Sudden increase in the blood pressure is, of course, significant. The body weight should be routinely recorded. Marked increase in weight is one of the earliest indications of the failure of kidney function. The patient should be instructed in dietetic and eliminative treatment. This usually means a salt-free diet containing 85 Gm. protein. She should be instructed as to danger signals, such as marked edema, headaches, pain in the epigastrium, and decrease

in the amount of urine. In the presence of increasing severity of symptoms, one must consider the termination of the pregnancy by the most conservative means.

The treatment of convulsive toxemia (eclampsia): The treatment of eclampsia is not satisfactory, as is shown by the large number of varying treatments suggested. Radical treatment should not mean surgical treatment only, but treatment of all cases by one method, whether by immediate cesarean section or medical management. The treatment of eclampsia is considered as surgical and medical.

Surgical treatment: Immediate termination of pregnancy, either by cesarean section, hysterotomy, or forcible delivery from below. The mortality rate is so high that the method is rapidly falling into disuse.

Medical treatment: The well-known method of Stroganoff recommends the use of large amounts of morphine and chloral hydrate. Venesection is used early. Delivery from below under chloroform anesthesia is advised. The Rotunda Hospital regime consists of colonic irrigation, starvation, and the parenteral use of 1 per cent sodium bicarbonate solution. Morphine and venesection are also used and labor induced after three days. Temple treatment: Dehydration is accomplished by repeated spinal drainage and magnesium sulphate given intravenously, by mouth or by rectum. Hypertonic glucose is given intravenously.

Veratrum Viride or its derivatives have been used in the treatment of eclampsia since the middle of the 17th century. This drug may have been misused in its early application, which may have accounted for the high mortality coincidental with its use. Zinke and, more recently, Bryant of Cincinnati have reported good results from its use.

At the John Gaston Hospital a variety of treatments for eclampsia was used during the three year period of 1938-1940. During this time there were 5,053 deliveries, of which 70 were eclamptic patients. Of these 15 died, giving a mortality of 21.4 per cent. During the three year period of 1942-44, there were 5,769 deliveries. Of these 70 patients had eclampsia, and 10 of them died, which shows a gross mortality of 14.3 per cent. In the latter period Veratrum Viride, or its derivative, veratrone, was used.

The object of this treatment is to control the convulsions by decreasing the blood pressure and to promote the excretion of toxins by diuresis. The blood pressure is reduced by the use of an antispasmodic drug, namely, veratrone, and diuresis is promoted by the

use of hypertonic glucose, using 500 c.c. of 20 per cent solution. Veratrone causes a dilatation of arterioles but is also a powerful cardiac depressant. The dosage must be adjusted to meet the individual case. As it is short acting, it must be administered every 15 or 20 minutes as necessary. The undesirable effects of the drug are in too rapidly lowering the blood pressure, slowing the heart, and the attendant vomiting. Although the mortality of our eclamptic patients has been decreased, it is clear that more improvement is necessary.

One of the few indisputable factors that has been observed in this condition is the reduction in urine volume. As oliguria increases, the patient's condition deteriorates, and with the appearance of anuria, death is certain. This has already been explained on the basis of vasospasm. All methods of treatment have as their aim the relaxation of the small blood vessels. The Stroganoff regime of using large amounts of drugs, such as morphine, had its success in the fact that the patient was not subjected to major surgery while in a desperate condition and that the drug given depressed the cerebral cortex basal ganglia, and finally the medulla, to the extent that the spasticity of small blood vessels was finally relaxed, and, as urine output increased, the patient naturally improved. This principle was directly applied in the use of veratrone, which has been discussed. We are now interested in other means of relaxing vasospasm, and recently in selected cases there has been considerable success from the use of caudal analgesia.

CASE 1. Mrs. F. M. H., aged 28, a gravida I, was six months pregnant. There was a sudden gain in weight, followed a few days later by a rapid rise in blood pressure from 120/78 to 170/110, and 4-plus albumin in the urine. She was admitted to the hospital on Sept. 8, 1945, at which time the blood pressure had increased to 204/120. The most important measure in treatment was continuous caudal analgesia for 9 hours, which reduced the blood pressure to 134/110, but it returned to 180/120 on the following day. She was kept in the hospital on a low salt diet until September 22, at which time her condition became acute. The blood pressure arose to 220/130, and she had a severe convulsion. A rapid and sustained pulse of 130 made us consider this to be the severe type of eclampsia. It may be that giving large volumes of hypertonic fluid intravenously to an already weakened heart might hasten cardiac failure with pulmonary edema. One cat unit of a digitalis preparation was given intravenously per hour for 4 doses and thereafter every 6 hours for 4 doses. There was practically no response in urinary excretion from giving 500 c.c. of 20 per cent glucose solution in the vein. A continuous caudal analgesia was given for 14 hours, using 340 c.c. of 1.5 per cent Metycaine. The blood pressure fell from 185/148 to 115/90. Four hundred c.c. of urine was excreted. Patient regained consciousness and was in good condition.

It was recognized that improvement was temporary, and with no hope of emptying the uterus from below, an abdominal hysterotomy was done. Al-

though the blood pressure rose the next day, with the reappearance of albumin in the urine, the patient made an otherwise uneventful recovery, and when seen for her follow-up examination six weeks after delivery, the blood pressure was 146/96 and the general condition satisfactory.

Caudal analgesia in this case took effect up to the diaphragm, relieving vasospasm to the kidney areas, which resulted in an increase in urinary output. It is not claimed that the treatment of eclampsia has been found, but it is clear that where no previous kidney damage existed, the relaxation of vasospasm was highly beneficial. It is probable that a series of selected eclamptic patients will be treated similarly, and we hope to be able to make a further report in the future.

It has also been noted that the serum proteins may be very important in eclampsia.

CASE 2. Mrs. C. B., aged 18, gravida I, was admitted to the hospital July 8, 1945, for convulsions. The blood pressure was 200/150, with albuminuria, and she was in deep coma. The urinary output was very unsatisfactory, and edema was severe. There was little response from 500 c.c. of 20 per cent glucose intravenously. On July 10, it was clear that pulmonary edema was developing, and we considered the patient to be in a dying condition. A serum protein determination showed total proteins of 3.5 Gm. per 100 c.c. of serum. A total of 2,000 c.c. of blood plasma was given slowly and in divided doses during the next 2½ days. Urinary excretion increased. On July 18, total proteins were 6.0, and the patient gradually recovered.

It is not claimed that low serum proteins are necessarily a part of the picture of eclampsia, or that giving blood plasma is the treatment, but it is evident that in severe nutritional deficiencies and in the presence of low serum protein values, it is highly beneficial to bring the serum protein level back to normal in order that the fluid elements of the blood may be better retained in the vascular system. We hope to make further reports on this problem. At the present time we shall continue to recommend the combined method of treatment as was suggested by Dr. W. J. Dieckman, as follows:

General treatment: The patient is placed in a quiet, darkened room. Constant observation is necessary to prevent injury to the tongue, falling out of bed, and aspiration of vomitus or drowning from the occasional excessive pulmonary secretions. A mouth gag (clothespin, toothbrush) and tracheal catheter should be at hand. A catheter should be kept in the bladder until the patient is definitely improving. The temperature, pulse, and respiratory rate, urine volume, and blood pressure should be taken every two hours until the patient is conscious and improving. The period is then lengthened to four hours and later the intervals are increased still further. The patient's condition as to the number of convulsions,

the degree of coma, the quality of the pulse, difficulty in breathing, cyanosis, etc., should be noted.

Convulsions: Since all drugs in the doses necessary to control the convulsions are toxic, we prefer to use several simultaneously. Smaller amounts are, therefore, required, and the undesirable effects of each are minimized. We prefer magnesium sulphate and luminal sodium. If the case is protracted, chloral hydrate is used.

Morphine: One-fourth grain is given on admission and repeated at hourly intervals until the convulsions are controlled or the respirations drop to 10 per minute.

Magnesium sulphate: Ten cubic centimeters of a 25 per cent, or equivalent amounts of a 50 per cent, solution are injected, and 5 c.c. of the former solution are given after each convulsion, or until a total of 30 c.c. has been given.

Luminal sodium: Three-tenths gram (5 gr.) is injected subcutaneously and if necessary repeated in 12 to 24 hours.

Chloral hydrate: Two grams (30 gr.) are given in 100 c.c. of starch water (tablespoonful of starch to 100 c.c.) by rectum and repeated as necessary.

Digitalis: Rucker suggests the use of half a cat unit to lessen the chances of occurrence of pulmonary edema.

Elimination: A soapsuds enema is used and should be repeated until a satisfactory bowel movement has been obtained. If the patient is vomiting, the stomach is emptied with a nasal tube, but no cathartic is injected. Sodium sulphate (Glauber's salt), 30 to 45 Gm., is a safer purgative than magnesium sulphate.

Hypertension: The blood pressure, at least in part, is compensatory. All sedative drugs lower it, but the barbiturates and chloral hydrate are the most efficacious or prevent further increases.

Renal and cerebral signs: The oliguria or anuria, coma, fever, tachycardia, and edema are treated with injections of hypertonic glucose solution. Five hundred to 1,000 c.c. of a 20 per cent solution are injected over a period of 30 to 50 minutes and repeated every 6 to 8 hours as is necessary. The temperature of the solution at the needle point must be 100° F. This can be achieved by immersing at least three feet of the rubber tubing in water at a temperature of 104 to 106° F. Within four hours after the injection, the urine volume should equal at least 60 per cent of the

volume injected. Sufficient glucose solution is given to insure a urinary output of at least 30 c.c. per hour and should be continued during the postpartum period until the normal diuresis begins. If the 20 per cent concentration does not produce a diuresis, 500 to 800 c.c. of a 30 per cent solution is used. If there is an anasarca, or if there are symptoms of cardiac failure, 300 to 500 c.c. of a 30 per cent, or 100 to 200 c.c. of a 50 per cent, solution is given. We have never seen any injurious effects if the glucose solution is properly prepared and administered.

Pregnancy: While the various procedures outlined above are being carried out, the period of gestation, size of fetus, and irritability of the uterus are being determined. No attempt to start labor or terminate the pregnancy should be instituted until the eclampsia is under control, which usually requires 6 to 10 hours.

Antepartum and intrapartum eclampsia: If there is cephalopelvic disproportion, a cesarean section should be performed when the eclampsia is under control. This presupposes that the case is not actually or potentially infected.

Severe cases of more than 35 weeks' gestation: The cervix will be uneffaced, firm, and closed. Pregnancy should be terminated according to existing conditions.

Mild case: Any period of pregnancy—No operative procedure should be carried out. Labor should be induced only if the cervix is soft and partly dilated. If the patient is not delivered, she must be kept under close observation until the pregnancy is terminated, because if the eclampsia recurs, it is usually of the severe type and is quite often fatal.

Postpartum eclampsia: The treatment is entirely medical.

Posteclamptic treatment: If the patient is conscious, or as soon as the stomach is emptying itself, as indicated by a failure to aspirate water which has been previously injected, a 10 per cent solution of Karo syrup is injected through the nasal tube at hourly intervals. The initial amount is 50 c.c. and is increased by 50 c.c. amounts up to the patient's tolerance (usually 200 to 300 c.c.). These feedings are continued until the patient can take the eclamptic diet which consists of water, fruit juices, and fruit. The fluid balance must be watched carefully and kept negative.

Miscellaneous procedures: The blood pressure should be watched carefully. Occasionally it drops before delivery and may require

treatment. After delivery a 10 to 15 pound weight (sandbag) should be placed on the abdomen and a tight binder applied for 8 to 12 hours to prevent vasomotor collapse. If the latter occurs, ephedrin (¾ gr.) every 2 to 4 hours, hypertonic glucose solution, and occasionally blood transfusions, are necessary. Atropine in doses of 1/100 gr. should be given if there are many rales. Up to 1/30 gr. should be given within one hour if pulmonary edema occurs. Oxygen by nasal catheter is used if the patient is cyanotic.

Anesthesia: Operative procedures can be performed quite often without any additional anesthesia because of the large amounts of narcotics already given. If additional anesthesia is necessary, local infiltration with novocaine solution is preferable. Where caudal analgesia has been used to lower the blood pressure and to relieve vasoconstriction, thereby encouraging an increase in urinary output, it is sometimes desirable to empty the uterus from above or below, depending upon existing conditions, while the analgesia is still effective.

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THE RETURNING SURGEON

In his speech on "The New South," Georgia's famous orator, Henry W. Grady, told of the Confederate soldier returning home after defeat at Appomattox as "ragged, half-starved, heavy-hearted, enfeebled by want and wounds, having fought to exhaustion." No such picture as this can be drawn for the American surgeon coming back from victory in Germany and Japan. There were fatalities and permanent disabilities among them, but the great majority are now at home, in good physical and mental condition, ready to assume the practice of their profession in a country not devastated by war. While some may discover that a proportion of their patients have fallen into other hands, most war surgeons will find a substantial nucleus of patients eagerly awaiting their return. New patients, too, attracted by the doctor's record and prestige, will seek his services.

Now may be a good time to begin the specialty which the veteran was considering before entering the war. His war experience may not have afforded opportunity for developing a specialty, but if he is determined to limit his practice the present seems a logical interval in which to make the change, and devote a few months to a preparatory course in some medical center.

Should he decide to begin activities at the same old stand he may learn that the place has disappeared or some other practitioner occupies it. On the other hand, a friend or relative fortunately may

have saved an office for him. It is gratifying to record that at least one large office building performed this praiseworthy service for its tenants gone to war.

Judging by what happened to the medical veterans returning from World War I, it may be safely predicted that the large majority of the surgeons of the late war who left home and practice to help win the conflict will not suffer professionally. In the main their patriotic sacrifice will be appreciated by their patients and the public at large, as well it should be. Whether or not their diagnostic acumen and surgical skill are improved, they met dangers and had experiences and adventures which will stimulate and inspire them throughout life. Certainly they are not to be discouraged, and may face the future with cheerfulness and confidence.

Today, however, a dark cloud casts its ominous shadow over the practice of medicine, both for the veteran and the stay-at-home surgeon. It is the possibility of socialized medicine, a deadly machine which affords a poor welcome to our returning surgeons. God grant that this rival of the destructive atomic bomb be not allowed to snatch from our veterans the methods of practice employed so successfully by the profession through many generations, and God grant that the sick of the nation be not subjected to a pernicious system of practice as designed by politicians and their ignorant and untrained accomplices.

FRANK K. BOLAND, M. D.

BOOK REVIEWS

The Editors of THE SOUTHERN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted without solicitation.*

COSMETICS AND DERMATITIS. By *Louis Schwartz,* M.D., and *Samuel M. Peck,* M.D., of the U. S. Public Health Service. 190 pages, with 20 illustrations. Price $4.00. New York, 1946: Paul B. Hoeber, Inc.

This book, although of special interest to the dermatologist and allergist, will also be of value to the industrial surgeon, particularly the chapters on occupational dermatitis and cleansers, including soaps and other detergents used on the skin.

The word "cosmetics" in the title is used broadly, including astringents, soaps, hand lotions, dentifrices, hair dyes and depilatories, lipsticks, nail polish, sun tan preparations, perfumes, deodorants, and powders. Sample formulas of most types of cosmetics are given. With such information available, one is better able to trace down the offending agent.

Several chapters devoted to diagnosis, methods of patch testing and treatment are very helpful.

Unfortunately, the illustrations, for the most part, did not print well. This, however, does not materially detract from the real value of this book as an excellent reference.

P. H. N.